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SECTION OF PEDIATRICS

TUESDAY AFTERNOON, JUNE 11, 1929

THE Section of Pediatrics of the Massachusetts Medical Society, held in the Copley-Plaza Hotel, Boston, convened at two-thirty o'clock, Dr. Charles A. Sparrow, the Chairman, presiding.

CHAIRMAN SPARROW: With the help of Dr. Garland, I have prepared a program which we hope will interest you.

The first paper this afternoon is "Dehydration," by Dr. James L. Gamble, of Brookline.

DEHYDRATION*

BY JAMES L. GAMBLE, M.D.†

THE purpose of this paper is to present a brief summary of several experimental attempts by the author and co-workers to learn the mechanism of the extensive loss of body fluids which so often accompanies severe disturbances of gastro-intestinal function.

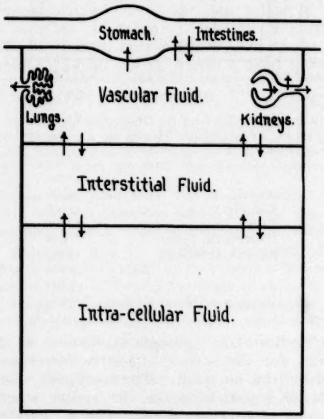

FIGURE 1. Representing roughly relationships of body fluid compartments.

The term dehydration implies a change in body fluid volume. Our knowledge of the physiological mechanism which controls body fluid volume is extremely fragmentary. In attempting to investigate changes in volume which disease sometimes causes, the basis of our thinking must therefore be, to a certain extent, hypothetical. In our studies we have used very simple and unpretentious premises; but we cannot, of course, be sure that they are correct.

The crude diagram, presented in Figure 1, will, perhaps, serve to bring at least the outlines of the body fluid situation into view. Water enters the body from the small intestine and is then distributed in three compartments; the vascular compartment, a space between the blood and the tissue cells, and inside the cells. Water leaves the body by way of the lungs, kidneys, skin, and, under normal circumstances, to only a slight extent in the feces. If these compartments had rigid walls, a flow of water through them at a stationary volume would not seem remarkable. Actually, however, their walls are elastically movable within wide limits, so that volume control must mean the operation of a very complicated mechanism of regulation. Obviously the chief locus of control of water removal from the body is the kidney. But the kidney is chiefly concerned in maintaining a correct concentration of substances in the blood plasma and we are, at least for the present, unable to discern a direct rôle in the regulation of plasma volume. There are, of course, many factors behind the kidney concerned in the regulation of volume in these several compartments. There is, for instance, the large mechanical factor consisting in the hydrostatic pressure produced by the work of the heart against elastic peripheral resistance. Also, the semi-permeable membranes separating these compartments produce particular osmotic pressure situations

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†For record and address of author see "This Week's Issue," page 957.

which have a great deal to do with the movement of water from the intracellular space back to the vascular space. But we must admit that the factors in control of volume, although evidently numerous and of complicated interplay, are as yet only vaguely visible.

I wish, however, to call your attention to the buffer or reservoir aspect of the middle body fluid compartment, especially from the point of view of sustaining, under varying circumstances, the normal volume of the blood plasma. The large accumulation of water which we call edema is localized here with the result that the blood plasma remains undiluted; and also, in all probability, cell volume is undisturbed. In the reverse situation, when circumstances tend to dehydrate the blood plasma, this reservoir provides a protracted defense of plasma volume. This extensive adjustability of the capacity of the interstitial body fluid compartment is an excellent example of the defensive type of physiological mechanism for which Cannon[1] has offered the term "homeostatic".

Now, although we know so very little about the factors in volume control, there is a basal proposition as regards physiological volume which can be accepted as at least approximately dependable and which provides a very useful premise for investigation of abnormal changes in volume; namely, that volume is determined by the total quantity of substances in solution according to the apparently very important physiological requirement for a nearly stationary concentration of substances. For instance, in edema fluid the concentration of substances is closely the same as in interstitial fluid when the volume of this fluid is normal. And we find that, when dehydrating circumstances have greatly reduced the volume of the blood plasma, the concentration of substances present remains near the normal value. So that, apparently, we should regard a change in volume as an adjustment in defense of the normal level of concentration of substances. In other words, an increase in volume must be the result of a retention of substances and a decrease in volume must be explained in terms of a loss of substances from the body.

This is fairly obvious, although not necessarily correct, reasoning, and constitutes one of the premises which we have used. According to it, dehydration is an incomplete term since it describes only a loss of water and does not indicate the primary event on which the loss of water depends, namely, a loss of substances. There is here the important corollary that dehydration cannot be repaired by water alone; in other words, in order to restore volume we must replace the lost substances along with water. Ross and the author[2] were at pains to demonstrate this point by a direct experiment. We found that the survival period of a dog, dehydrated as a result of obstruction of the pylorus, could not be appreciably prolonged by intra-peritoneal injections of isotonic glucose solution. Presumably the glucose thus given is rapidly oxidized so that this procedure amounts to giving water alone. If, however, the same quantity of physiological salt solution be used, the dog can, as was first shown by Hartwell[3], be kept alive for several weeks. Glucose solution is for several reasons, which will presently be considered, an important agent in the treatment of dehydration but it should be remembered that it does not directly restore volume and that for this chief requirement of the situation we must depend on salt solution.

Having placed the emphasis on a loss of substances as the primary event in dehydration, how can we explain this loss? In the case of dehydration accompanying severe disturbances of gastro-intestinal function, we have used what would seem to be the most apparent hypothesis; namely, that vomiting and diarrhoea cause a continued loss of gastro-intestinal secretions and thereby produce a progressive withdrawal from the blood plasma of the materials and accompanying water used in the construction of these secretions.

This hypothesis requires quantitative support. That is, is this inner circulation between the blood plasma and the gastro-intestinal tract of a magnitude sufficient to explain the extensive loss of water and substances which occurs in severe dehydration? So far as water is concerned, we have evidence that it easily is. Table I, taken from a review article on water metabolism by Rowntree[4] presents estimations of daily volume for the several digestive secretions as produced by an adult. The combined volumes of these secretions make the rather startling value of about eight litres. This is more than twice the volume of the blood plasma. Evidently these data suit the conception that circumstances interfering with reabsorption of these secretions will explain a rapid withdrawal of water from the vascular compartment.

TABLE I

ESTIMATIONS OF VOLUME OF DIGESTIVE SECRETIONS PRODUCED IN TWENTY-FOUR HOURS BY AN ADULT. CITED FROM ROWNTREE.

	cc.
Saliva	1500
Gastric juice	2500
Bile	400
Pancreatic juice	600
Succus entericus	3000
	8000
Volume of blood plasma	3500

Having postulated a loss of substances as the cause of the water loss, we are here required to demonstrate that these secretions contain the substances which support body fluid volume. Curiously, there are almost no data in the literature descriptive of the inorganic composition of the digestive fluids. McIver and the author[5]

have been for some time collecting information on this point. The gist of the information which we have obtained is presented by means of the diagrams in Figure 2. There are two secretions entering the stomach; the acid gastric juice which contains chloride ion, hydrogen ion, and fixed base in the relative amounts indicated in the diagram, and the mucous secretion of the stomach which is alkaline in reaction because it contains more of fixed base than of chloride ion, the excess of base being present as bicarbonate. The specimens of this secretion were obtained from an isolated pouch constructed in the pyloric antrum where there is no production of acid juice. Pancreatic juice contains fixed base in large excess over chloride ion. Secretions drained by a fistula from an upper jejunal loop contain base and chloride ion at about the concentrations found in blood plasma. This is also true of bile taken from the hepatic duct. The total quantity of substances in each of these secretions is, as the heights of the diagrams indicate, closely the same as is found in the blood plasma. These data demonstrate that the gastrointestinal secretions contain plasma materials in amounts sufficient to explain their volume, and that, when these secretions are lost, there occurs a parallel withdrawal from the blood of total substances and of water. We also learn from these studies that the materials taken from the blood plasma are, chiefly, fixed base and chloride ion. The fixed base in these secretions we have found to be composed almost entirely of sodium. The extremely simple therapeutic agent, physiological salt solution, is thus indicated for the repair of dehydration (a).

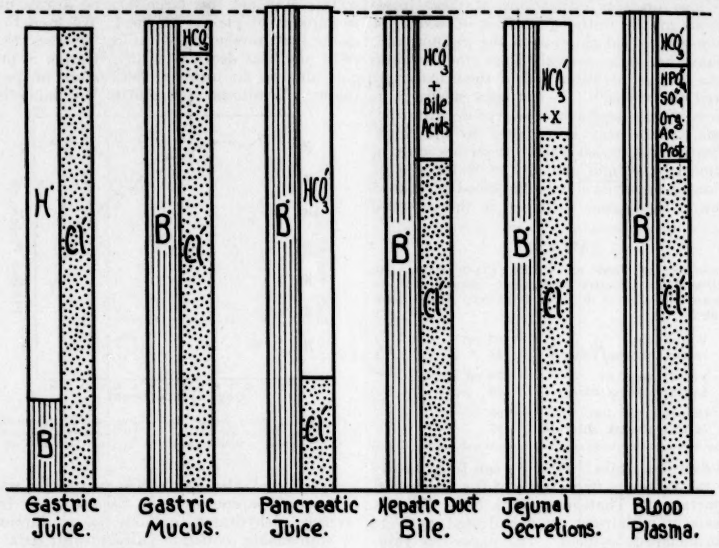

FIGURE 2. Ionic composition of digestive secretions. The chief factors are fixed base (B'), chloride ion (Cl'), and bicarbonate ion (HCO'_3). In gastric juice, hydrogen ion (H') is also a large factor. The fixed base value in these secretions and also in the blood plasma is composed almost entirely of sodium. Cations are placed in the left hand and anions in the right hand columns of the diagrams. Pancreatic juice data from dog. Other data from cats.

(a) The following comments may be added to this brief description of the composition of the digestive secretions. The gastric juice diagram was constructed from measurements obtained from specimens taken from an isolated pouch in the fundus. The specimens must have contained a slight admixture of gastric mucus which might entirely account for the fixed base value found. The composition of bile taken from the gall bladder[6] is not represented in Figure 2. There occurs in the gall bladder a remarkable concentration of fixed base to far above the blood plasma level and also an almost complete removal of chloride ion. With regard to the bicarbonate factor in the upper intestinal secretions it should be pointed out that bicarbonate ion does not represent material lost from the plasma. Bicarbonate is present in the body fluids to such extent as fixed base exceeds the sum of the other acid ions and derives from the free carbonic acid which is sustained at a fixed level throughout the organism.

By way of actual quantitative evidence of the extent of the loss of water and of materials which may occur in the presence of circumstances preventing reabsorption of secretions, data which McIver and the author[6] obtained from rabbits are given in Table II. These experiments consisted in obstructing the pylorus by ligature, then sacrificing the animal as near the end of the survival period as could be guessed, and measuring the amounts of water, of sodium, and of chloride ion which had entered the stomach. The rabbit is a uniquely suitable animal for this

experiment because it does not vomit. The data thus obtained describe the extent to which water and materials may be withdrawn from the plasma by loss of gastric secretions alone. The table also presents estimations of these losses in terms of the initial quantities of water, of sodium and of chloride ion in the plasma, and, as may be seen, several times the original plasma content of water and of these materials entered the stomach. It has been shown that, in conditions producing severe dehydration, plasma volume may be reduced by one-third. Evidently this measurement describes only a fraction of the total quantity of water and of substances withdrawn from the blood plasma.

How is the plasma sustained in the presence

TABLE II

ESTIMATIONS OF LOSS OF WATER, FIXED BASE, AND CHLORIDE ION, FOLLOWING PYLORIC OBSTRUCTION, IN TERMS OF ORIGINAL PLASMA CONTENT. DATA FROM RABBITS.

Water lost	203	cc.
Initial plasma volume	83	"
Fixed base lost	270	cc. 0.1N.
Initial plasma base	140	"
Chloride ion lost	309	"
Initial plasma chloride	85	"

of so drastic a drain? Here we can fully appreciate the defensive importance of the interstitial compartment. That interstitial body fluid is extensively withdrawn in dehydrated patients is conspicuously evident. The paper-like thinness of the skin and, in infants, the deeply sunken fontanelles are, indeed, the outstanding physical signs of the condition. McIver and the author[7] have recently demonstrated experimentally that the blood plasma may be completely sustained, over a considerable period of progressive dehydration, by water and materials derived from interstitial body fluids. In this experiment dehydration was produced at a gradual rate in a dog by completely draining away, by means of a fistula, the external secretion of the pancreas. Pancreatic juice, as we have seen, contains fixed base, which is almost entirely sodium, at about the same concentration as does the blood plasma. The animal was fed boiled minced meat which contained only a slight amount of sodium. Water intake was not restricted. Dehydration due to the progressive sodium deficit is indicated in Figure 3 by the loss of body weight which, as may be seen, begins at the outset of the survival period. The plasma protein measurements also given in the figure provide evidence that a normal plasma volume was sustained for about ten days. The limit of support from the interstitial reservoir is then apparently passed and plasma volume falls rapidly as indicated by the sharp rise in protein concentration.

We may now ask regarding changes in the third and innermost body fluid compartment which contains intracellular water, or, in other words, the water of protoplasm, here we have probably four-fifths of the water content of the body. May not this large lake be drawn upon in support of plasma volume? We used to assume that severe dehydration involves tissue cells, and that death from dehydration is probably due to an ultimate desiccation of protoplasm. Measurements made in this laboratory

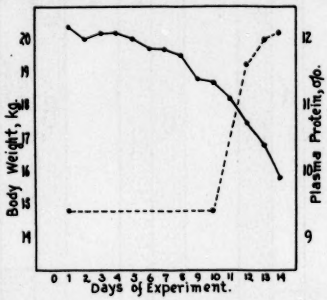

FIGURE 3. Body weight and plasma protein measurements from a dog during survival period following establishment of a fistula which drained away the external secretion of the pancreas.

by Peck and also by McKhann of the water content of parenchymatous tissues taken from severely dehydrated animals show, however, no appreciable reduction (unpublished data).

Obviously it is enormously to the advantage of the organism to be able to retain its cell water. On the basis of dissections of the chemical anatomy of the several body fluids, we have been able to devise an explanation of this defense of the texture of protoplasm. The diagrams in Figure 4 describe the essential structural factors in the composition of blood plasma, of cerebro-spinal fluid, and of muscle juice. Cerebro-spinal fluid may be taken as representative of interstitial body fluid and muscle juice as an example of intra-cellular fluid. It will be seen at once in the diagrams that in blood plasma and in cerebro-spinal fluid sodium ($Na^{\bullet}$) constitutes nearly all of the base and that chloride ion (Cl') is the chief acid factor. We have noted that these are the substances withdrawn from the plasma when reabsorption of gastro-intestinal secretions is interfered with. The suitability of interstitial fluid for replacement of blood plasma losses is thus apparent. Looking at the muscle juice diagram we note that most of the base is potassium ($K^{\bullet}$) and that most of the acid value is phosphate ($HPO4''$) (b). Now potassium cannot replace sodium in the plasma nor can phosphate substitute for chloride ion, and, according to the premise which we are using, water without substances will not support volume. In other words, an expenditure

of water and materials from the intra-cellular compartment, with consequent disruption of the structure of protoplasm would be futile from the point of view of supporting the blood plasma. Apparently, the logic of the situation prevails.

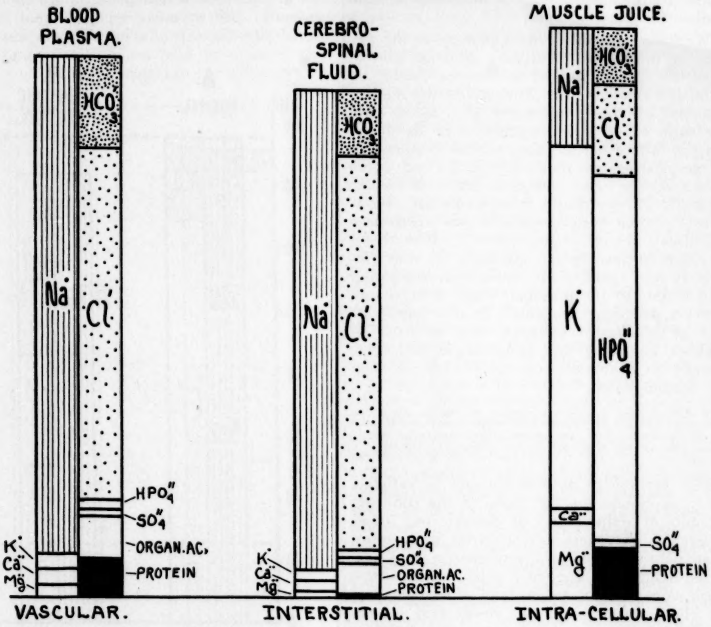

FIGURE 4. Ionic composition of the body fluids. The diagrams are constructed from average normal concentration values for the individual factors. The cations are superimposed in the left hand and the anions in the right hand column. The diagrams represent, as is actually the case, a structure composed, not of salts, but of individually sustained concentrations of ions. The exact acid-base equivalence, indicated by the equal heights of the two columns, is obtained by the adjustability of the bicarbonate ion concentration (HCO'_3). The cerebro-spinal fluid data are those of Hamilton[8]. The muscle juice diagram is constructed from a few unpublished measurements and is probably only roughly descriptive.

(b) It is quite probable that the intra-cellular water of muscle tissue contains no sodium. The diagram in Figure A is constructed from measurements obtained from the juice pressed out from a specimen of fresh muscle. This material must to some extent contain interstitial fluid, a relatively small amount of which owing to its high sodium content would explain the concentration of sodium represented in the diagram. In the case of the red blood cells, it has been definitely shown that their water of protoplasm does not contain sodium.

These data and considerations persuaded us that dehydration is limited to loss of water from the interstitial and from the vascular compartments. An amendment to this view has, however, recently been added by the results of experiments carried out in this laboratory by Drake and McKhann. Using rats, dehydrated as a result of experimental obstruction at the pylorus, they found that, although the per cent. content of water in muscle tissue is not appreciably lowered, there does occur a considerable reduction of the total mass of muscle tissue in the rat. This can only mean a destruction in toto of muscle protoplasm producing a corresponding disappearance of water and of substances together. When we consider the circumstances under which dehydration occurs, a consumption to some extent of protoplasm is not a surprising event; the factor of fasting is always present in such situations and might alone explain it. By way of indicating the extent of the oxidation of body protein during simple fasting, it may here be mentioned that Ross, Tisdall and the author[9] estimated, on the basis of nitrogen excretion, a loss of seven pounds of protoplasm by an epileptic child during a two weeks' fast undergone as a therapeutic measure. But water released by destruction of protoplasm is probably not available in support of plasma volume because of the unsuitableness of the accompanying inorganic substances and is, we may surmise, removed along with these substances in the urine. We have experiments under way which, it is

hoped, will definitely demonstrate this point. Admitting then an appreciable consumption of body protoplasm in situations producing dehydration, we can nevertheless retain our conception that a desiccation of protoplasm is prevented by the unsuitability of intra-cellular substances for plasma repair.

We have dealt so far with changes in the volume of interstitial fluid and of blood plasma resulting from a loss of substances, chiefly sodium and chloride ion. Now, unless the relative amounts of these substances lost agree with the ratio of their concentrations in the blood plasma, alteration of the normal composition of the plasma must inevitably occur; and whenever a change in an inorganic factor or factors occurs it always causes a compensating alteration of plasma bicarbonate and this adjustment produces a change of reaction (c). It is, therefore, not surprising that clinically we very frequently find changes of reaction accompanying dehydration of the plasma. These changes are, however, curiously variable. In diarrhoeal disease in infants for instance, there is often an acidosis, but sometimes an alkalosis, and occasionally no appreciable change of reaction from the normal value.

(c) For a more extensive discussion of the factors determining the bicarbonate concentration in the blood plasma than can be undertaken here, the reader is referred to an earlier paper, Acidosis and Alkalosis, by the author and published in this journal[10].

This variability of reaction can be satisfactorily explained on the basis of the differing amounts of sodium and of chloride ion found in gastric secretions as compared with upper intestinal secretions. A loss of gastric juice will, as the diagram in Figure 2 indicates, withdraw much more of chloride ion than of sodium from the blood plasma, with the result that plasma bicarbonate must increase; the rôle of bicarbonate must increase; the rôle of bicarbonate being to maintain the acid and basic factors in exact equivalence. The causal relationship of vomiting to alkalosis has been thoroughly shown. By way of illustration the diagrams in Figure 5 are presented. The right hand one is constructed from measurements obtained by Ross and the author[2] from the blood plasma of a patient with a malignant obstruction at the pylorus who had vomited continuously for several days, and shows a very extensive loss of plasma chloride and the resulting huge increase in bicarbonate. Pancreatic juice, on the other hand, contains much more of sodium than of chloride ion so that we might expect continued loss of this secretion to cause reduction of the plasma bicarbonate. McIver and the author[7] confirmed this surmise by the already cited experiment in which the external secretion of the pancreas was drained away through a fistula. The measurements obtained from blood plasma taken from the dog before establishing the fistula and on the fourteenth day of the survival period are represented graphically in Figure 6. We may therefore dependably conclude that vomiting tends to cause alkalosis and diarrhoea acidosis; and that the reaction of the blood plasma in diarrhoeal disease of infants where both symptoms are present will depend on which is predominant. But we must remember that in most clinical situations producing dehydration other

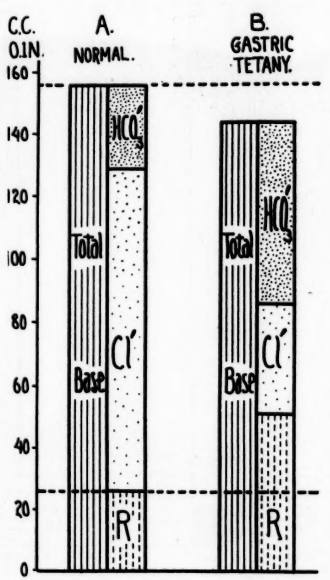

FIGURE 5. Alkalosis caused by loss of gastric secretions. The diagrams are constructed from measurements of the chief acid-base factors in the blood plasma. Note in the gastric tetany diagram the huge extension of the bicarbonate ion concentration (HCO'_3) due to the large loss of chloride ion (Cl') from the plasma.

factors affecting the composition of the blood plasma may be present. There is, for instance, not infrequently a ketosis of varying degree. And it is important to keep in mind the often extensive impairment of renal function which is a consequence of severe dehydration. We are indebted to Hartman[11] for placing a proper emphasis on this factor and for thoroughly describing the various distortions of plasma structure caused by a failing control of the concentrations of plasma substances by the kidney.

Besides these changes of blood plasma composition with resultant alteration of reaction, there occurs as a result of reduction in plasma volume, extensive changes in the physical properties of the blood. There is for instance an increase in viscosity with the result that volume flow may be enormously reduced. Utheim[12] was

able to measure in dehydrated infants a tenfold reduction in the volume flow of blood. Dehydration of its plasma must therefore cause a progressive impairment of the functions of the blood, and this to our minds has always seemed a sufficient explanation for failure of physiological processes and the ultimate death of the organism. It has long been noted that the symptoms accompanying dehydration resemble those of so-called toxemias; there is alternating drowsiness and irritability and, finally, convulsions and coma. The term Intoxication offered by Finkelstein is still used to describe the severe phase of nutritional failure in infants produced by diarrhoeal disease, the outstanding physical feature of which is dehydration. The assumption that the symptoms accompanying dehydration of gastro-intestinal origin are caused by absorption of a toxic substance is still very generally used. This additional hypothesis seems to us unnecessary, especially since these alleged evidences of toxemia, even when extremely severe, can usually be made to disappear simply by restoring body fluid volume.

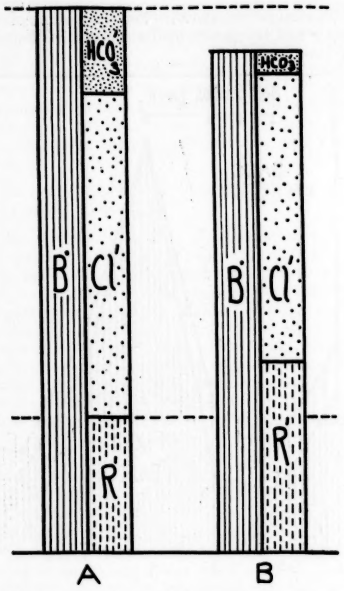

FIGURE 6. Acidosis caused by continued loss of the external secretion of the pancreas. Diagram A presents the measurements of the chief acid-base factors in blood plasma taken from a dog before establishing a fistula draining away the external secretion of the pancreas. Diagram B shows the change in these factors found on the 14th day of the survival period. Note the reduction of the bicarbonate (HCO'_3) value due, chiefly, to loss of plasma fixed base (B').

The therapeutic measures indicated by the above discussion of pathogenesis are extremely simple and may be very briefly presented. We find that dehydration accompanying disturbances of gastro-intestinal function is the result of loss of body fluid materials and that this loss is almost entirely composed of sodium and of chloride ion. Energetic administration of physiological sodium chloride solution is, therefore, the basal step in an attempt to restore body fluid volume. To be sure, sodium and chloride ion individually have usually been lost to a differing extent and by giving salt solution we supply these materials in equal amounts. But, fortunately for simplicity of therapeutic procedure, we can depend on the kidney to deal separately with sodium and chloride ion according to their respective deficits in the blood plasma. The validity of this assumption is demonstrated by data given graphically in Figure 7, taken from an experiment by Ross and the author[2]. This ability of the kidney to correctly reëstablish the sodium and chloride levels in the plasma, provided water and sodium chloride are abundantly supplied, also incidentally brings about a restoration of the plasma bicarbonate to its normal value, so that we are not required to attempt direct repair of the accompanying alkalosis or acidosis, except when ketosis is a factor in the latter.

The reparative adequacy of salt solution as regards both the volume and the reaction of the blood plasma and interstitial fluids depends, however, on the presence of an alert renal function. We have noted that progressive disability of the kidney is a consequence of dehydration. The essential factor apparently is simply a lack of available water. A second and very important item in the treatment of dehydration is, therefore, to supply a surplus of water with the purpose of restoring renal function. Water given as salt solution tends to be retained because it contains the substances required for rebuilding body fluid volume. Glucose solution, however, since it does not contain these "structural" substances should excellently serve the purpose of bringing the kidney into active and accurate operation. And there are other services which glucose solution provides. If given intravenously as a ten per cent. solution, it very greatly hastens the absorption of subcutaneously delivered salt solution. This effect is probably due to the temporary hypertonicity of the plasma caused by injecting a glucose solution of this concentration. Glucose will also demolish an accumulation of ketone acids in the plasma if this abnormal circumstance is present. Moreover, glucose, by its protein sparing effect, should greatly reduce the rate at which protoplasm is being destroyed. These several considerations support the use of intravenous glucose solution as an extremely important accessory measure in the treatment of severe dehydration.

The intravenous injection of bicarbonate solu-

tion has been recommended in those instances where an accompanying acidosis is of severe degree. This is a debatable point. We are inclined to believe that this additional procedure is rarely, if ever, necessary (d).

(d) By way of indicating the quantity of these solutions which dehydrated patients may require, the customary procedure at the Children's Hospital in the treatment of severely dehydrated infants may be cited here. Physiological salt solution is given subcutaneously or by intraperitoneal injection to the extent of about 30 cc. per kilogram of body weight, and 15 to 20 cc. of 10 per cent. glucose solution per kilogram is then given intravenously. It is often necessary to repeat this procedure once or even twice at intervals of about eight hours, in order to obtain satisfactory replenishment of the body fluids as indicated by physical appearance and, especially, by the reëstablishment of urinary function.

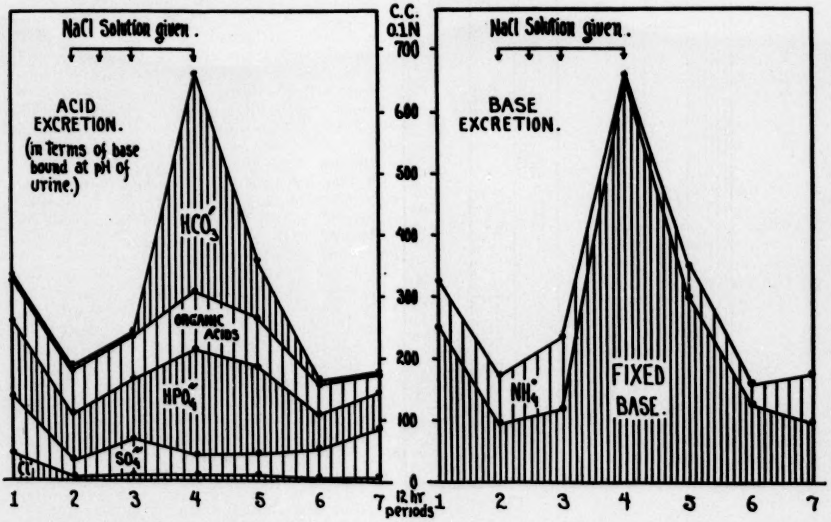

FIGURE 7. Illustrating the control by the kidney of the excretion of sodium and of chloride ion individually following injections of sodium chloride solution.

The diagrams are constructed from measurements of acid and of base factors in successive 12-hour specimens of urine taken by catheter from a dog dehydrated by an obstruction placed at the pylorus. Loss of gastric secretions produces a larger deficit of chloride ion (Cl') than of sodium (Na^+) in the body fluids, so that, when sodium chloride solution is then given abundantly, a much larger surplus of sodium than of chloride ion will present for excretion in urine. The data here given demonstrate that this situation is correctly managed by the kidney. In the left hand diagram it may be noted that, following the intra-peritoneal injections of salt solution, the quantity of chloride entering the urine remains minute whereas there soon occurs a large increase in the excretion of sodium as shown by the fixed base measurements in the right hand diagram. Referring again to the left hand diagram the mechanism of the conveyance of the surplus sodium into the urine is evident in the corresponding extension of the excretion of bicarbonate ion.

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DISCUSSION

CHAIRMAN SPARROW: The discussion of this paper will be opened by Dr. Harold L. Higgins, of Boston.

DR. HAROLD L. HIGGINS, Boston: *Mr. Chairman*—Dr. Gamble's paper is surely a masterpiece. He has summed up the situation very well and it is rather difficult to find anything to disagree with or to add.

Obviously, a great many of his deductions have been the result of clinical observations, for which he has found scientific background. When we were working together in Baltimore about ten years ago, we used indiscriminately normal salt solution and 5 per cent. glucose solution for intra-peritoneal injection with the children who were dehydrated as a result of summer diarrhea. It appeared that the normal salt solution was more efficacious clinically in treating the chil-

dren during the acute stages of dehydration. We formerly used salt solution for two or three injections and then, if further were needed, we thought that glucose was preferable.

A number of pediatric clinics are using Ringer's solution, which contains not only sodium chloride but potassium, calcium and phosphate, etc., in dehydration rather than the usual normal salt (sodium chloride) solution. I would like to ask Dr. Gamble whether he thinks that the physiological Ringer solution is preferable to a normal salt solution in treating the dehydrated child.

As regards the loss of fluids, it would seem that when the cells lose their fluids, they have a tendency to die, and when we get sufficient destruction of body cells, if those body cells are vital cells, then the dehydration will prove fatal.

CHAIRMAN SPARROW: This paper is now open for general discussion.

DR. EUGENE C. PECK, New London, Conn.: Dr. Gamble, perhaps you remember that we had some rabbits with diarrhea in the laboratory just before you came. Those rabbits showed the loss of water from a sample of skin taken by rapidly dissecting off the skin down to the superficial fascia of the muscle, that is the fascia covering the muscle and giving water content on the skin, muscle and liver. We found practically no change in the muscle and liver at a time when there was a very pronounced change, about 9 or 10 per cent. The water in the skin was greatly diminished. Do you suppose that water is intra- or inter-cellular water? Perhaps any cellular tissue there contains that water, but not actually in cells.

Then there is another question I would like to ask you. In two different hospitals I have seen the operating room have a good deal of difficulty in getting first-class, clear, sterile glucose for intravenous use. A week ago I set up a porcelain filter intending to try it out to see. Will that be a practical means of preparing a glucose solution—simply dissolve our C. P. glucose in distilled water and run it through a previously sterilized porcelain filter as we do certain serological products? Do you know if that has been done, or whether it is worth trying?

DR. JAMES L. GAMBLE, Brookline: As regards the suggestion that Ringer's solution be used instead of isotonic sodium chloride solution, we have felt that, insomuch as the material lost is composed almost entirely of sodium and chloride ion, replacement is probably adequately obtained by giving salt solution.

I am much interested by Dr. Peck's description of his experiments with rabbits and especially his finding of no appreciable loss of water from the parenchymatous tissues. The water lost from the skin I should think without question was interstitial water. The lower layers of the skin being loosely areolar provide a large depot for interstitial fluid.

We have not had experience with untoward reactions following injections of glucose solution. I am not competent to offer an opinion as to the effectiveness of filtration as a means of avoiding such reactions. I should like to refer this question to Dr. Blackfan, whose experience is much wider than mine.

DR. K. D. BLACKFAN, Brookline: I don't recall having had any difficulty with the preparation of glucose solution. There are one or two preparations on the market that can be added to sterile water or distilled water, as the case may be, which are very effective in use.

The main consideration, I think, in the preparation of solutions for intravenous or intraperitoneal use is that they shall be prepared freshly and that they shall be sterilized promptly, because of the so-called salt fever reaction which has been shown, I think, quite definitely to follow the use of solutions which have been sterilized after the solutions have been made up and kept for some time.

There is another very interesting point also. You may get reaction from the use of intravenous solutions if the rubber tubing isn't properly prepared. Fresh rubber tubing contains substances which do cause very sharp febrile reactions and sometimes more serious reactions, and it is rather important that new rubber tubing should be first permitted to stay in a weak alkaline solution for twenty-four hours, or overnight, and then a weak acid solution for overnight, and then in cold, distilled water for another period of time.

I think the preparation of the distilled water and the rubber tubing are the chief factors, of course using pure sodium chloride and pure glucose.

CHAIRMAN SPARROW: The second paper on the program is "Brucella Abortus Infection of Milk," by Dr. Merrill J. King, Mount McGregor, New York, Director of the Laboratory of the Metropolitan Life Insurance Company Sanatorium.

BRUCELLA ABORTUS INFECTION OF MILK*

BY MERRILL J. KING, M.D.†

IN July, 1926, one of the maids employed at the Sanatorium at Mount McGregor became ill. She had a low grade fever and her blood count showed a leukopenia with a relative lymphocytosis. Her Widal, however, was negative and cultures of blood and feces were negative for B. typhosus. At this time, case reports of undulant fever were just beginning to find their way into the literature. An agglutination test was therefore done with this patient's serum and a titre of 480 with Br. abortus was obtained. This was our introduction, three years ago, to the problems of undulant fever and this case initiated some of the studies referred to in this paper.

In discussing undulant fever, three possible etiological factors must be considered,—Brucella melitensis, Br. abortus of porcine origin and Br. abortus of bovine origin. In the southern and southwestern part of the United States, goats are infected with Br. melitensis and typical cases of Malta fever have been reported from this section of the country by Craig[1], Gentry and Ferenbaugh[2], and Yount and Looney[3]. In 1927 Watkins and Lake[4], reported an epidemic of Malta fever in Arizona which arose from drinking goats' milk infected with Br. melitensis. It is possible that our dairy industry may be jeopardized from this source but there seems little need of apprehension from this quarter at present.

The "Bacillus abortus" of Bang and the "Micrococcus melitensis" of Bruce undoubtedly belong to the same bacterial species and it is only with considerable difficulty that these strains may be differentiated. Br. abortus itself may again be subdivided into bovine and porcine strains, both of which cause undulant fever in man. The great majority of cases reported have been of bovine origin. Carpenter and King[5] in an analysis of 155 reported cases found that only seven of these had direct contact with swine or had been on farms where abortion disease in swine had occurred, while 109 of these patients had a history of drinking raw milk from which, in the majority of cases, Br. abortus was isolated. Fortunately abortion disease in swine is extremely rare in the eastern United States—only recently Theobald Smith[6] recorded an outbreak of infectious abortion in a herd of swine in New Jersey which is probably the first reported in the east. Hardy[7] in Iowa, where swine raising is a leading industry and where contagious abortion among swine is not uncommon, found 11 of 83 cases of undulant fever to be of porcine origin. McAlpine and Slanetz in Connecticut are doing some excellent work attempting to devise methods, other than serological, whereby the bovine and porcine strains of Br. abortus can be differentiated. In our work at Mount McGregor there has been no evidence of porcine infection and all of the cultures isolated by us have been of the bovine type. As far as I can learn only one culture of Br. abortus has been isolated from swine in the last ten years in New York State. It is difficult to determine whether abortion disease spreads from cattle to swine or from swine to cattle. However infectious abortion in cattle was described many years before it was reported in swine and in most parts of our country the disease is common in cattle and uncommon in swine. In a few instances, cases of undulant fever have occurred in meat inspectors or butchers who have been in contact with swine but possible contact with infected cows that have aborted, or with infected milk, has not always been considered. Without doubt a small percentage of the cases of undulant fever occurring in the United States may be traced to wound infections.

Abortion disease is widespread in this country and it has been recognized for at least two centuries in Europe. McAlpine and Mickle[8] found 90% of the herds of Connecticut to be infected, Kern[9] reports that at least 86% of the herds of Pennsylvania are infected and Belyea[10] speaks of abortus infections as being "widespread in the cattle in the Pacific Coast States". Many of the herds that we have tested in New York, New Jersey, Massachusetts and Vermont have been infected.

In 1911, Schroeder and Cotton[11] described a "new pathogenic bacterium in milk" which was in all probability Brucella abortus. In examining milk for tubercle bacilli by means of guinea pig inoculation, they obtained lesions suggestive of tuberculosis although no acid-fast bacilli could be found. Their description of the organism and its incidence strongly suggest Br. abortus. In 1912 Smith and Fabyan[12] pointed out that Br. abortus from milk when inoculated into guinea pigs produced a disease with well-defined lesions from which the specific organisms could be isolated. Since that time extensive studies have been made on the prevalence of Br. abortus in milk. Carpenter found 20% of the samples of raw milk of 67 towns and three cities in New York to be infected and 18% of the milk of 50 dairies of one city contained the bacilli. At Mount McGregor we found 54% of our herd of 151 cows were infected and although our milk was certified, 29.2% of these infected cows were eliminating the abortus organisms in their milk. Fleischner and Meyer[13] as a result

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†For record and address of author see "This Week's Issue," page 957.

of guinea pig inoculations with certified milk, concluded that "Br. abortus, for all practical purposes, is always present in certified milk produced in the San Francisco Bay region". Since certification does not, as yet, consider abortus infection in herds it is not surprising that we have repeatedly isolated the Br. abortus organisms from certified milk of different dairies. The menace to public health of drinking raw infected milk is apparent and will be considered later in the paper.

The incidence of abortion disease in cattle and the virulence of the abortus organisms are extremely variable in different herds. Br. abortus is discharged from the uterus and mammary gland of cows and at the time of abortion the placenta and uterine exudate are teeming with organisms. A large percentage of cows that abort show this infection in the colostrum but it may soon disappear after normal milk is secreted. However, 20 to 80% of this group continue to eliminate the organisms in their milk throughout their lifetime. We have found that infected cows may at times give non-infected milk. Some of the cows in our herd have always given us positive milk cultures but with many of the infected cows, at least one milk sample has failed to infect guinea pigs. This suggests that the organisms may not be continuously discharged into the milk and that reliance should not be placed upon a single negative report. The greatest number of abortus organisms are found in the cream content of milk, where they have been carried by the fat globules.

As a rule, comparatively few (20 to 500) abortus organisms can be cultivated from one cubic centimeter of milk. Since abortus organisms are usually less virulent than melitensis and since there are usually so few in cows' milk, it seems logical that human infection is not as common from Br. abortus as it is from Br. melitensis. It has been experimentally proven by Fleischner, Vecki, Shaw and Meyer[14] that one or two feedings of 1:1000th of the amount necessary to cause an infection with Br. abortus is a sufficient dose of Br. melitensis to infect monkeys.

Within the last three years considerable data have been accumulated suggesting the prevalence of human abortus infection. In fact, as early as 1913, Larson and Sedgwick[15] in complement-fixation tests using an abortus antigen on sera of 425 children who had drunk raw milk found antibodies for this organism in 17%. In another group of children, who had drunk milk from a dairy in which there had been no abortion among the cows, they did not obtain a single positive reaction. In 1917 de Forest[16] published a series of cases of twelve abortions in women in which circumstantial evidence pointed to milk infection. The following table indicates the findings of the majority of investigators who by serological tests on human sera have attempted to ascertain the occurrence of Br. abortus infection in man. The chief source of the material studied has been from blood samples sent to public health laboratories for the Wassermann test; some examinations have been made upon sera from normal adults and children who have drunk raw milk. The source of the serum, the serum dilutions considered significant, the antigen used and the procedure vary. All of these factors may produce variations in the final results.

					No.	%	
Larson and Sedgwick	1913	Minn.	425	children—raw milk	73	17	
Ramsey	1915	Minn.	116	children	7	6	
Williams and Kolmer	1917	Penn.	62	women having aborted	0	0	
Fiel	1922	Italy	98	patients—tuberculosis	11	11.2	
Evans	1925	Va. and D. C.	500	patients—various diseases	59	11.8	
Carpenter	1926	N. Y.	20	patients—undiagnosed fevers	5	25	
Carpenter and Chapman	1928	N. Y.	4,050	Wassermanns	296	7.3	
Hardenbergh	1928	Minn.	4,766	routine medical examinations	201	4.2	
Giordano and Abelson	1928	Ind.	1,000	patients—various diseases	59	5.9	
Kristensen	1928	Denmark	1,177	Widal bloods	89	7.6	
King	1928	Mass.	690	patients—tuberculosis—pasteurized milk	0	0	
Hardy	1928	Iowa	783	Widal bloods	68	8.6	
McAlpine and Mickle	1928	Conn.	10,102	Wassermanns	64	0.6	(1:100)
King and Caldwell	1929	N. Y.	599	patients—various diseases—infected milk	82	13.9	

The data of King and Caldwell[17] were based on sanatorium patients who were drinking certified milk. Bacteriological studies were then in progress on the sanatorium herd and, as I have before stated, 24 of the 151 cows were shown to be eliminating Br. abortus in their milk. During a seven months' period, after the infected cows had been removed from the herd and while guinea pig inoculations showed the milk of each remaining cow to be free from Br. abortus, 270 new individuals were tested and the serum from none of these possessed specific agglutinins for Br. abortus. The data indicate that when living abortus organisms were present in the milk, specific agglutinins appeared promptly in the blood serum of the patients, but that when the milk was abortus-free there was no infection of the patients. Further corroboration was obtained by the results of agglutination tests on the bloods of patients of

three state tuberculosis sanatoria in Massachusetts where only pasteurized milk was used. Six hundred and ninety sera were thus examined and none agglutinated Br. abortus.

Further evidence of the danger of feeding raw infected milk is shown by the situation at a small college in Indiana where four students were found to be suffering from undulant fever. The college maintained its own dairy of twenty cows and supplied raw milk to the student body. Agglutination tests on the blood sera of the cows showed that 50% of the animals were infected with contagious abortion. Three of the animals were eliminating Br. abortus in their milk. Pasteurization of the milk was instituted and no new cases of undulant fever have appeared.

McAlpine and Mickle[18] report six cases in a state institution in Connecticut. All had drunk raw milk from a herd which was badly infected with contagious abortion.

Although the experience at Mount McGregor, in Indiana, and in Connecticut show that human beings may be infected from ingestion of cows' milk containing Br. abortus, you may have observed that these organisms must be but slightly pathogenic, for only a small percentage of the individuals consuming the infected milk in these institutions showed evidence of infection.

A recent report of 209 cases of undulant fever in Denmark comes from Kristensen[19] of Professor Bang's laboratory. In 37 of these patients the source of infection was not known, 69 were infected from drinking milk, 60 had been drinking infected milk and handling infected cattle, while 43 patients were infected from contact with infected cattle. Kristensen succeeded in culturing Br. abortus from 18 of 29 of the patients. In none of these cases was any evidence found of swine infection nor of Br. melitensis from goats.

The age incidence in undulant fever is apparently greatest in young adults from 15 to 35 years. Males have been reported to be more commonly infected than females but this may be explained by the fact that as a rule, men drink more milk and cream and they are more likely to be associated with the handling of infected animals and carcasses on farms and in slaughter house... Farmers and people of rural districts who usually drink raw milk have been more frequently infected than city dwellers. Our experience would also indicate that patients suffering from other diseases, particularly those who drink considerable infected milk, are especially susceptible to infection with Br. abortus. There have been instances of the infection of laboratory workers handling cultures of Br. abortus—and this has become almost the rule when bacteriologists manipulate Br. melitensis. One bacteriologist became accidentally infected with Br. abortus by cutting his finger with a scalpel while autopsying a guinea pig which had been injected with abortus-infected milk. Apparently the majority of patients are infected by ingestion of the organisms; however, we must not overlook the dangers of infection from contact with the abortion-products of cows and hogs and the handling of infected carcasses in abattoirs.

Both Br. melitensis and Br. abortus produce three distinct types of undulant fever. The severe, text-book picture, the milder form resembling an attack of influenza and the mild ambulatory type which is common with both Br. abortus and Br. melitensis although it is seldom recognized. Shaw[20] cultured true melitensis from 10 of 22 apparently healthy Maltese dock-hands; in fact the ambulatory type is said to be the commonest form of undulant fever caused by drinking goats' milk in Malta.

The period of incubation is usually from 10 to 14 days in man. There are no pathognomonic symptoms of undulant fever. The severe cases usually show chills, fever and sweats, headaches, joint and muscle pains, extreme weakness, with variable gastro-intestinal symptoms and loss of appetite. Skin rashes and small petechial spots occasionally occur in the skin, and blurring of vision has been noted. The European writers usually stress mild respiratory symptoms, although this has not been commonly noticed in cases in America. These severe cases may be ill with remissions and exacerbations for a period of many months. The mild influenza-like cases are indistinguishable clinically from influenza. It is in these cases that laboratory tests are especially valuable in differential diagnosis. Undoubtedly the extreme variability of symptoms and resemblance to other commonly suspected diseases account for the frequent failure to diagnose undulant fever.

Among other diseases to be considered in the differential diagnosis are influenza, typhoid fever, malaria, tuberculosis, endocarditis, rheumatism, infectious mononucleosis and tularemia. Infrequent complications are orchitis, spondylitis and hydrarthrosis—Dr. Harold Amoss* of Johns Hopkins has cultured Br. abortus from fluid of the knee joint. Carpenter* has cultured abortus from one human fetus which was aborted at the end of the fourth month. We were unable to isolate Br. abortus from 49 pairs of tonsils injected into guinea pigs, while Carpenter* found the organisms in 7 out of 55 pairs of tonsils.

In the diagnosis of undulant fever, laboratory confirmation is essential, there being three important diagnostic procedures:

(1) *Cultural studies:*—For blood cultures, at least 5 to 10 cc. of blood are planted in liver vitamin broth or in liver vitamin agar. Bovine Br. abortus requires an atmosphere of about 10% carbon dioxide for optimal growth and

*Personal communications to the author.

the cultures should not be considered negative before 14 to 21 days have elapsed. Even under these conditions it is often very difficult or even impossible to culture Br. abortus from the circulating blood. Br. abortus may sometimes be cultured from the urine of patients suffering from undulant fever. It is advisable to inject blood and urine samples into guinea pigs; occasionally Br. abortus may be recovered in this manner when cultures have failed.

(2) *Serum reactions:*—Blood serum agglutination tests should be made on both heated and unheated specimens, using a carefully prepared abortus antigen which is known to be sensitive. We routinely use serum dilutions of 1:15, 1:45, 1:135 and 1:405 in our agglutination tests. The occurrence of the pro-agglutinoid zone phenomenon is not at all infrequent and for this reason it is important that dilutions at least through 1:400 are made. The work of Carpenter* and of Evans[21] has demonstrated that human beings may have a Br. abortus bacteremia without possessing agglutinins in their blood serum; the former has four times cultured Br. abortus and the latter was once successful in culturing Br. abortus from the blood of patients whose sera did not agglutinate specific antigens. Tramanto[22] failed to find agglutinins in the sera of 9 of 65 confirmed cases of abortus infection. We were twice successful in isolating Br. abortus from the milk of a cow before agglutinins appeared in her blood serum. Because of this variable antigenic property of certain strains of Br. abortus, it is unfair to specify any given serum titre as diagnostic of undulant fever. Then too, Br. abortus agglutinins disappear very slowly from sera and a titre of 1:405 may persist for two years after apparent recovery from the disease. We have found that positive complement fixation usually accompanies abortus infection, except in the early stages of the disease, and the serum may continue to fix complement for many months after the infection has occurred. This agrees with the findings of Kristensen.

(3) *Skin tests:*—Skin tests for diagnosis were introduced by Et. Burnett[23] and he finds them more reliable than the agglutination test. Giordano and Abelson[24] and several other investigators rely on the intradermal reaction and we have had encouraging results by injecting 0.1 to 0.05 cc. of killed Br. abortus into the dermis of guinea pigs and monkeys. Usually within six hours a well-defined area of reddening and edema appears and within 24 hours there is a definite induration and in many animals sloughing occurs. We would advise against the universal adoption of this diagnostic measure until further animal experimentation has been made. The intradermal use of abortin with animals has not given us reliable results.

The treatment of undulant fever is largely symptomatic although several drugs have been used and may be of some service. Mercurochrome has been used in a few cases with variable results but definitely controlled evidence of its value is lacking. Br. abortus vaccines are relied upon by some and the immune serum from convalescent patients has been administered intravenously. An immune cow serum is now on the market for the treatment of human beings infected with Br. abortus.

You may well ask why so few cases of undulant fever have been recognized in infants and young children. The fact that children are apparently not so susceptible as adults cannot as yet be explained. We all do know, however, that for some reason infants are quite immune to many infections and there may be some "group-immunity" in children which protects them against infection with Br. abortus. I am aware of but four cases of abortus infection which have occurred in children under six years of age. The youngest of these is of particular interest and has not yet been reported in the literature. A patient of Dr. Enid Crump[25] two years old, was believed to be suffering from rheumatic fever until a chance agglutination showed that the child's serum agglutinated Br. abortus antigen through a dilution of 1:1280. She had been transfused with blood from her father and from an uncle and it was later found that they both had abortus agglutinins in their blood sera. The physicians in charge concluded that all three had been infected from drinking cows' milk and that the child had been infected with Br. abortus previous to the transfusion. You will recall that Larson and Sedgwick found 17% of 425 children who had drunk raw milk, and Ramsey, 6% of 116 children, to show blood serum evidence of Br. abortus infection.

Veterinarians are well aware of the fact that many calves which are fed abortus-infected milk do not develop evidence of disease or agglutinins in their sera. Calves that do develop agglutinins do not harbor the organisms in their tissues after the source of infection has been removed; only one per cent retain agglutinins until the time of first conception. This is in marked contrast to the permanency of contagious abortion infection occurring in mature cattle. Simms and Miller[26] demonstrated that calves which have ingested an abundance of infected milk prior to weaning are immune and they were not able to demonstrate the development of agglutinins in sera of these calves subsequent to their feeding. Huddleson[27] found that serum antibodies specific against abortus develop occasionally in calves as a result of ingesting naturally infected milk, providing their intestinal tracts are made permeable by an existing enteritis. Calves at birth, even when borne by infected mothers, never have agglutinins for Br. abortus in their blood serum.

I mention this information relative to the immunity of calves to Br. abortus in an effort to

*Personal communication to the author.

find some logical explanation for a possible immunity in infants and children for undulant fever. It seems likely, however, that unrecognized undulant fever does occur in infants and young children, but that they are not as susceptible to the infection as adults. Are we too rash in suggesting that some of the babies with undiagnosed fevers might be found to be suffering from undulant fever, should their blood be examined for evidence of Br. abortus infection?

SUMMARY

(1) Contagious abortion is widely prevalent in cattle throughout the United States.

(2) Approximately 20% of raw market milk is infected with Brucella abortus.

(3) Brucella abortus of both bovine and porcine origin is pathogenic for man.

(4) It is impossible to specify any given serum agglutinin titre as diagnostic of undulant fever.

(5) Ingestion of infected milk and direct contact with infected animals are the chief causes of undulant fever.

(6) Either universal pasteurization of milk or a new code of certification of milk is essential in the control of undulant fever.

(7) That undulant fever is a public health problem is indicated by the increasing number of cases being reported.

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DISCUSSION

DR. LEWIS W. HILL, Boston: Dr. King has presented such an extremely clear and complete exposition of the subject that I can add but little.

I wish to impress upon the minds of this audience that undulant fever, once it is acquired, is a serious disease. It is no longer a medical curiosity; it is common enough now so that it must always be taken into consideration in the differential diagnosis of a long-continued obscure fever with a low white count.

How important is undulant fever as a public health problem, and how likely is anyone living in Massachusetts to acquire it? From various government reports it seems clear that there are few herds of cattle which are not infected with contagious abortion.

One must assume, therefore, that a considerable quantity of the raw milk consumed in this country contains the bacilli of undulant fever. Still, although a fair number of scattered cases have been reported, there has been no large epidemic, such as would be the case if milk were infected with typhoid bacilli or streptococci. One must conclude therefore that the abortus bacillus possesses a very low degree of infectivity for man, and for this reason, that while undulant fever is certainly of considerable importance from a public health viewpoint, its importance is by no means to be compared to that of such a disease as typhoid fever, diphtheria or scarlet fever.

In Massachusetts there have up to the present time been reported four cases of undulant fever, one in Worcester, one in a student at Smith College, Northampton, (not acquired in this state), one in a bacteriologist at the Children's Hospital, and one in a twelve year old boy in Newton, a patient of mine. From talking with various herd owners and veterinarians in Massachusetts one gathers the impression that contagious abortion is just as common here as it is in the states from which many cases of undulant fever have been reported. Massachusetts is not a dairy state, and a good many of the cases of undulant fever reported in other states have been in dairy workers. Furthermore it is essentially an urban state, and in most of the large cities a considerable portion of the milk consumed has been pasteurized. Perhaps these two factors may partly account for the small number of cases reported here.

One must conclude from the evidence that a considerable amount of the raw milk consumed in Massachusetts contains living abortus organisms, and that therefore anyone drinking raw milk does run a risk of contracting undulant fever. Owing to the relatively slight infectivity of these organisms for human beings, this risk is small. It is particularly small for infants, and as far as we know, to date no cases of undulant fever in infants have been reported.

On June 17, 1928 I was called to see an eleven year old boy in Newton who had had a fever for two days. On physical examination there was nothing to be found and he had no symptoms, other than feeling tired and listless. He has not been free from fever for one day from that day to this. He has never shown anything on physical examination, except a moderate degree of anemia, and has been remarkably free from symptoms save for occasional headaches

and sweats. His temperature has varied between 101 and 105: at present it is about 101 every night. He was kept in bed for the first few months of his illness, and for the last few months has been in bed two days a week, and up and around, indulging in a moderate amount of exercise the rest of the time.

When he was first taken sick his white blood count was 5400. Typhoid, malaria or tuberculosis were suspected, but were ruled out by repeated laboratory examinations. In July 1928 a specimen of his blood was found to agglutinate Bacillus Melitensis in addition of 1 to 80 and this was confirmed in a second test a few weeks later. Blood and urine cultures were negative. It seems certain that this boy has undulant fever. The source of infection is, however, impossible to determine: he had never come into contact with animals, and had never taken any raw milk.

Any disease which can disable a patient for a year, as it has done to this boy, is certainly a formidable one, and the time is not far distant when as much attention will be paid to the occurrence of contagious abortion in a dairy herd, as is now paid to tuberculosis.

During the reading of Dr. King's paper the reporter was called to report a meeting of the Council, and the remainder of the Pediatric Section was not reported.

The following officers were elected to serve for the ensuing year in the Section of Pediatrics:

Chairman: Dr. Orville R. Chadwell, Boston.
Secretary: Dr. Joseph Garland, Boston.

ANNOUNCEMENT OF RED CROSS ANNUAL ROLL CALL DATES

Announcement has just been made of the forthcoming Annual Roll Call of the American Red Cross, for membership during the coming year, to be held from Armistice Day to Thanksgiving Day, November 11 to 28, inclusive.

The response to this Roll Call directly reflect the degree of knowledge of what the Red Cross does each year in each community. Where a Red Cross Chapter, with the support of a strong membership, is conducting a public health nursing program in its community, is active in assistance to veterans, or carries out a program attracting volunteer activities among its people, thus giving the town or city an even wider representation in its functions than membership enrollment alone affords, the people generally understand just what their membership does for them and for their section.

Out of each dollar membership in the American Red Cross, fifty cents is retained by the Red Cross for expenditure in behalf of the community. In other words, all except fifty cents of the member's enrollment, regardless of amount except in the case of life or patron memberships, is spent in his own town, city, or county, as the case may be. Thus, a large Chapter enrollment means direct benefit to the community.

The fifty cents from membership enrollment which goes to National Headquarters is spent in national and international work. Each year thousands of dollars are expended by National Headquarters for relief in disasters for which no general appeal for funds is made to the public. In many instances annually, National Headquarters advances funds to local Chapters for emergency relief in their communities following disaster, in order that this work will not be delayed while a public subscription of relief funds is under way. Such help is only possible because of the support of a large membership. The increasing demands on the organization's personnel and funds in this field alone stresses the importance of a representative enrollment for the coming year.

Where Red Cross Chapters have raised in their jurisdictions large sums for relief of disaster, these expenditures have been in every case accounted for to the public by an audit by accountants of the United States Government.

The degree of participation of each member of the Red Cross in the work of the Chapter is to a large extent optional, but the member who enrolls even for a year has the satisfaction of knowing that he has given financial and moral support to his or her Red Cross, and to this extent is an active participant in its work everywhere. Join!

N. E. BRANCH OF AMERICAN UROLOGICAL ASSN.

MELANOTIC SARCOMA OF THE PENIS*

Report of a Case

BY FLETCHER H. COLBY, M.D.†

A MASSACHUSETTS shoe-worker, single and 48 years old, consulted Dr. George Gilbert Smith in February, 1929, for a sore on the penis. One and one-half years ago the patient first noticed a slightly elevated, circular area on the side of the glans. This was darkly pigmented from the beginning and slowly increased in size. The surface of the lesion developed into a blister which had broken a few weeks before he was seen. No history of trauma was given and there had been no application of irritating substances to the diseased area. Except for this the patient was well.

Examination of the right half of the glans penis showed a roughly circular area of black pigmentation. The edges were slightly puckered and sharply defined from the normal mucous membrane. The central portion was pink in color, slightly raised and a little nodular, with some induration. This portion was superficially ulcerated. No enlargement of the inguinal lymph nodes could be detected. General physical examination revealed no other abnormalities and the Wassermann report was negative.

On March 4, 1929, a partial amputation of the penis was performed by Dr. Smith. Although a more radical procedure was advised, this was all the patient was willing to submit to.

Convalescence was uneventful, the tissues healed rapidly, and the patient was discharged 10 days after operation.

The pathological report on the diseased portion of the glans was melanotic sarcoma.

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†For record and address of author see "This Week's Issue," page 957.

Primary sarcoma of the penis is relatively rare. Joelson in 1924 from Dr. Quinby's clinic, reported a case of fibrosarcoma arising from the penis with notes on thirty-five additional cases of sarcoma. They represented the total number he was able to collect from the literature. Only eight of these thirty-six tumors were melanosarcomata. The melanosarcomata occurred in men from 52 to 75 years of age. In two instances the growth had been present for as long as eight years; in one instance metastatic involvement of the regional lymph nodes was present as early as nine months after the appearance of the primary lesion. So the rate of growth of these tumors seems to vary considerably. One of the cases was untreated and died two years after onset with metastasis. In seven instances amputation of the penis was performed. Four of these seven cases died of extensive metastases within seven months. In the remaining three no details were given. Though metastasis was usually found in the regional lymph nodes, it is worthy of note that in one of these cases metastatic involvement of the liver alone was reported.

Although more slowly growing than other forms of sarcoma of the penis as shown by the duration of symptoms, the melanotic type is definitely malignant and may metastasize readily. Operative results in the past have been poor, however, six of the reported cases were treated before 1900, and it is possible that present surgical methods will produce better results.

SUTURE OF KIDNEY WOUNDS—EXPERIMENTAL*

BY CLYDE LEROY DEMING, M.D., F.A.C.S.†

THE suture of kidney wounds is as diversified as there are numbers of surgeons. As one visits the different Clinics, one often sees kidneys cut open from pole to pole, the wound approximated with large, transfixing sutures of various types or a large piece of fat interposed to control the bleeding. Many nephropexies are being done by heavy transfixing sutures. What is the future of these kidneys? Greater respect for the circulation of these operated kidneys should be demanded, and a more or less stable opinion formed as to the method of suture of these kidney wounds.

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†For record and address of author see "This Week's Issue," page 957.

Moore and Corbett have emphasized the following facts concerning their experimental work with kidney wounds: (1) operations on the kidney always destroy some kidney substance; (2) sectioning of the kidney did less harm than the suturing necessary to control the hemorrhage; (3) suture of the capsule alone was not sufficient to control hemorrhage and therefore dangerous; (4) the mattress suture placed transversely through the kidney destroys a great deal of kidney substance and the enlargement of the unoperated kidney, a short time after, was due to over-work and not to increase in kidney substance; (5) destruction of the kidney substance extends far beyond the field of operation; (6) the functional activity is somewhat reduced; and (7) interrupted sutures transfixing the kid-

ney at the pyramidal line and tied around the body of the kidney did the least damage and therefore is the method to be recommended.

Apparently from the study of the literature everyone who has done experimental work on suturing of the kidney has improved his own

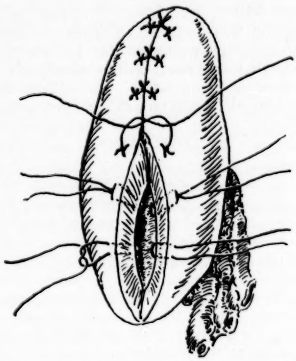

FIG. 1. Longitudinal nephrotomy showing method of suture with No. 00 chromic catgut. Suture placed close to line of incision passed from cortex to pelvis.

technique by it; but there still seems to be a chaos of ideas as to the methods of suture and the material used. The suturing of a nephrotomy wound aims: (1) to control the hemorrhage; (2) to avoid tissue damage by strangulation or tearing; and (3) to approximate the edges.

With these facts in mind, experimental work was conducted on normal, healthy dogs. Urine examinations and functional kidney tests were done before and after the operations. The dogs were operated upon under ether anaesthesia using aseptic technique. Various incisions and various types of sutures were used, usually both kidneys were operated on at the same time. The kidneys were allowed to remain for three or four months and then they were removed. Their arteriovenous circulations were irrigated with sodium citrate solution and then injected with a celluloid solution which gave a corrosion model of the exact size of the circulations. Histological sections were not made. This has been done by Goldstein and others but I doubt if this gives a true idea of blood vessel conservation. The width of the scar tissue does not give any idea as to the amount of blood vessel obstruction and destruction. It is assumed, and I think correctly, that when there is no obstruction to the ureter or to the kidney circulation, the function of a kidney is proportional to the amount of its circulation.

First, normal kidneys were injected so as to obtain a normal model. Then the kidneys were opened from pole to pole through the so-called bloodless line of Hyrtl and sutured by means of deep chronic catgut stitches, some placed in mattress form and others in Lambert's type to close the kidney wound and control the bleeding. The capsule was then sutured with superficial stitches of plain catgut.

The gross and corrosion model specimens showed marked shrinkage in size, a diminution in the number of vessels and a marked decrease in the number of glomerulae. A functional test in these cases was about two-thirds normal. It is quite evident from a large number of specimens that much kidney damage is done by the deep sutures.

The next series of kidneys were sutured by means of mattress sutures placed at a considerable distance from the cut surface. These sutures were passed down on one side, through to the pelvis, up on the opposite side and back again to the original side. These corrosion specimens show marked changes in the circulation not unlike the first series.

Complete transverse nephrotomies were then attempted and the wounds sutured by means of

a *b*

FIG. 2.
Sketches Showing Suture of Transverse Nephrotomy.
(*a*) sutures placed at a distance from incision; (*b*) sutures placed close to incision.

double "O" chromic mattress sutures on each side. One series was placed close to the cut edge and the other at some distance. The corrosion models show distinct difference between these two methods of suture. Those kidneys in which the sutures were placed close to the incision, show very little change in the corrosion model; while those kidneys with sutures placed at some distance from the cut edge, show deep grooves and destruction.

Angulated incisions at the suggestion of Marion are difficult to suture without destroying

some of the circulation at the angle. All of these corrosion models show grooves with some destruction of the circulation. Although the incision be carried in the mid line on to the kidney and then transversely through the bloodless zone, one frequently encounters vessels which bleed and a deep suture is required to control the hemorrhage.

When the incision is extended from the pyelotomy wound on to the lower pole of the

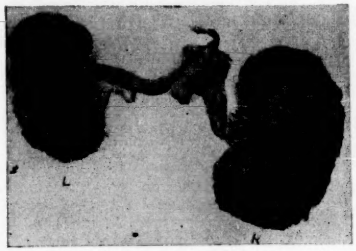

FIG. 3. Corrosion model of dog's kidneys in which complete longitudinal nephrotomies were done. L, result of sutures placed at a considerable distance from cut surface. R, result of suture proposed.

kidney in the mid line, one sees but little change in the gross specimen or in the corrosion model.

Complete end to end nephrotomies sutured with fine No. 00 chromic sutures placed close to the lines of incision in mattress form on each side, easily control the hemorrhage and destroy but little circulation.

Various types of incisions were made with wires and sharp knives and I am thoroughly convinced from the specimens, that wounds produced by sharp instruments and sutured with fine chromic sutures destroy the least tissue and conserve the greatest number of vessels. It is not difficult to place these fine sutures close to the lines of incision through the capsule to the pelvis and back again. These sutures readily control the hemorrhage and when tied with its opposite fellow, give a complete closure of the wound and there was no evidence of leakage in any of the experiments so far performed. All the animals were closed without drainage. None of them leaked any urine. None died of peritonitis and all recovered without any complications.

CONCLUSIONS

1. Deep transfixing sutures destroy and obstruct vessels of the kidney.
2. Angulated incisions are difficult to make and suture without cutting and ligating important vessels—by so doing obvious defects in circulation are demonstrated in the circulation models.
3. Kidney wounds can be sutured by mattress sutures of No. 00 chromic catgut passed from cortex to pelvis with little permanent damage to the circulation.
4. Complete longitudinal and transverse nephrotomies may be done with impunity if sutured by the method here described.

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A CASE OF RESECTION OF HALF OF A SOLITARY KIDNEY*

BY THOMAS N. HEPBURN, M.D., F.A.C.S.†

DR. THOMAS N. HEPBURN, Hartford: I feel very apologetic in coming before you this evening because on reaching Boston I found the slides of my case had been left out of my bag. I had expected to speak with the aid of the slides, but Dr. Graves wants me to go ahead without them.

The case I wish to report is resection of half a double kidney in a man who had had the other kidney removed. This man came on my Service at the Hartford Hospital four years ago in a very serious condition. He hadn't passed any urine for three days. He had acute pain in his right renal region and gave a history of having had his left kidney removed ten years before for stone. His N. P. N. was 88, his creatinin was six. His blood pressure was 130 over 90; his leucocytes were 17,000; temperature 103, pulse 110. He looked very ill, and was intensely nauseated, with dry tongue. I fortunately was able to get a catheter into his right ureter past an obstruction near the kidney, and pussy urine flowed. The x-ray showed that he had a double kidney with bifid pelvis on this side, and that the upper kidney was entirely filled with a large stone cast, protruding into the pelvis. Part of this stone had broken off and lodged at the junction of the ureters, obstructing the good lower kidney so that the man had an infected hydronephrosis. The catheter drained for 24 hours and then got plugged. I had to operate through a lumbar incision and remove the stone that was plugging the bifid pelvis. The man made a satisfactory recovery. He left the hospital in 28 days with a normal N. P. N. and creatinin and a phthalein output of 22 per cent.

I then advised him and his family that he should have a heminephrectomy done because I saw nothing but a repetition of this catastrophe. They declined, much to my delight; and he went home. I was called

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up again 48 days later and told that he was in the hospital. This time he had not passed urine for four days and he was practically in the same condition as before: leucocytes were 15,000, tongue dry, etc. His N. P. N. at that time was 95.

I got permission to do a radical operation and quickly operated through the old incision, removing the upper half of his kidney with the large stone cast and took out the stone that was plugging his bifid ureter. This man drained urine profusely into his wound for three days and then began to void normally and left the hospital in 29 days with a normal blood chemistry and phthalein output of 20 per cent.

When I was asked to present a case here I called up this man and had him come back to the hospital. Last week I cystoscoped him again and had pictures to show. The lower kidney pelvis is normal. The ureter is perfectly good, and his phenolsulphonephthalein output is 48 per cent. in two hours. He feels better than at any time.

Now the interesting part of this case is the functional result. I think we have very few detailed reports of such clinical material to study. I find that Judd has done a heminephrectomy in six cases reported in 1925. All of them had a second good kidney. In three of these cases he found that the function in the remaining half of the partially resected kidney became bad and he had to later remove the other half of the kidney.

Hugh Young reported a case in 1917 in which he did a heminephrectomy for almost the identical condition as in my case only there was no obstruction in the pelvis and there was a second good kidney. Young's case before operation showed a phthalein output of 20 per cent. from the diseased side and 30 per cent. from the normal side. Four months following his heminephrectomy the diseased side showed a phthalein output of five per cent. and the good kidney had taken on compensatory function.

I think the surgical principles involved in these cases are clear. If we have a good kidney on the other side we are not justified in doing a partial kidney resection. It is wiser to do a complete nephrectomy. On the other hand, if we have a single kidney with a pathological condition which gives us the slightest opportunity for resection, this case shows that by resection of that pathological portion the remaining good portion shows remarkable compensatory capacity.

I am very much interested in that large group of cases in which there is bilateral renal pathology and in which probably partial renal resection is indicated on both sides. Unfortunately I have no clinical material by which to know how those kidneys are going to react.

Technically I wanted to show a procedure which I thought might be a help to you. In reading the technique described, by those who describe partial resection, they are careful to say, "isolate your circulation first before you do your resection so as not to disturb the circulation of the good kidney." I can assure you that in a surgical kidney such as the one I have described, this would be a hazardous procedure. There is a marked perinephritis and congestion of the kidney. Your patient is in extremis and any attempt to identify the nutrient blood vessels to the good part of the kidney would be a slow and dangerous procedure. In the double kidney type there is a definite furrow between the two kidneys which gives us a good indication for their division. Both Judd and Young prefer to separate these two halves with the knife; and Judd lays emphasis on resecting a good margin of healthy kidney. Young describes in detail cutting a V out of the good kidney, and curetting the entered calices. He then closes the cut end of the good kidney with mattress suture so that capsule is brought to capsule.

Now I would like to show you some pictures similar to Dr. Deming's done by Braedel and published in Kelly and Burnham's work showing the circulation in double kidneys. The blood vessels in such kidneys do not cross from one to the other. I would suggest that instead of using a knife that you niche the capsule and use your finger to separate them by blind dissection. You will find that they separate like a prostate from the capsule. No calyx is entered. The hemorrhage is very slight. When separated you have your vascular supply definitely before you and you can tie.

I would like to say that I found very little oozing from the cut end of the good kidney, and what little there was, was controlled by a few mattress sutures. I did not bring the capsule together over the cut end, nor put fat up against it, but put a cigarette drain there. That kidney in spite of the ureterotomy drained through the wound only three days; and the urinary fistulas in all the other cases reported continued a number of weeks. There is no danger of breaking through a calyx with that method, while with a knife you are apt to cut through a calyx.

In conclusion the lesson to be drawn from this case is that there is an encouraging field for surgical resection of the diseased part of a kidney where there is only one kidney. If you have a good kidney on the other side, partial resection is useless. As a technical procedure, try using your finger to separate double kidneys, and do not bother with attempting to cover the raw end of the kidney with capsule or fat.

REMOVAL OF BILATERAL RENAL CALCULI*

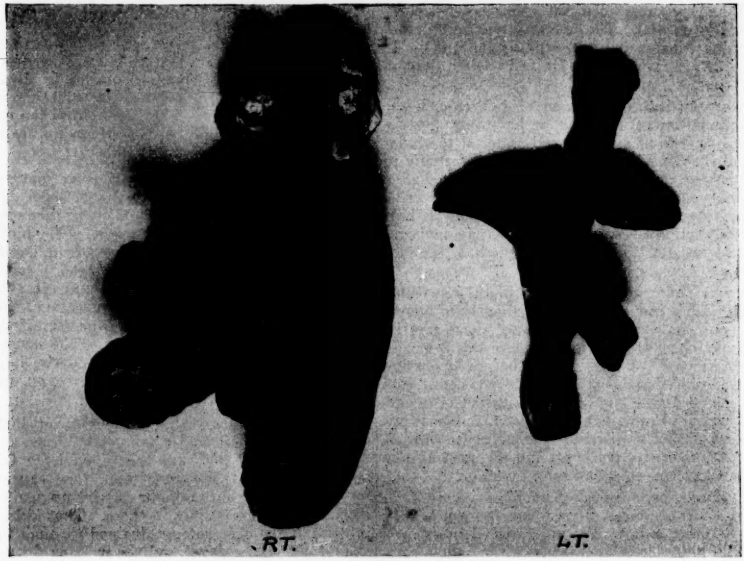
BY C. H. NEUSWANGER, M.D.†

I AM going to report briefly four cases which have come under my observation recently and have required operations upon both kidneys.

The first case, H. W., was a young man with multiple renal calculi on the right with an impacted right ureteral calculus. He also had multiple left renal calculi with colon infection in both kidneys. When I first saw the patient he had a complete obstruction of the right ureter. The right ureteral stone was within a few cm. of the kidney, and this was removed together with the stones in the kidney at the first operation. The patient was operated on six months later when the stones in the left kidney were removed.

The second case, W. C., was a very similar case of bilateral renal calculi without any obstruction which did not present any unusual difficulties, and the stones were removed at separate operations.

The third case, M. D., was a young woman thirty years of age who for two years complained of pain in both sides and this was accompanied by burning and frequency. A cystoscopic examination revealed a large stone in the right kidney without infection. The left kidney showed a slight hydronephrosis with numerous pus cells and colon bacilli. The function was normal in both kidneys. The patient was operated upon and the stone in the right kidney removed. She recovered from this operation with no complications, but six months later she developed severe pain in the left side with burning and frequency. An examination at this time showed the left kidney to be functionless. It contained a large amount of pus with tubercle bacilli. The right kidney was negative for pus cells and excreted 22% of phthalein in thirty minutes. The blood chemistry was normal. An x-ray of the patient's chest showed bilateral pulmonary tuberculosis. The patient was operated upon and the left kidney removed under spinal anesthesia using 100 milligrams of Neocain. The patient made an uneventful recovery from this operation.



Stones Removed in Case V. B.

The interesting points about this case are that the patient did not develop tuberculosis in the right kidney when the stone was removed. That the patient stood the second operation very well under spinal anesthesia and this did not aggravate the lung condition.

The fourth case, Mrs. V. B., a Lithuanian housewife, aged fifty-two, complained of pain in both sides for two years, during which time she had been passing blood in the urine and having burning and frequency, otherwise the patient had no complaints and did not give any history of previous urinary symptoms.

The abdominal examination revealed a large palpable mass in the right lower quadrant which was

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slightly tender on pressure. The abdomen was obese but otherwise there were no areas of tenderness or masses felt.

CYSTOSCOPIC EXAMINATION

The cystoscope was introduced with difficulty and the bladder contained 40 c.c. of cloudy urine. Both ureters were catheterized without difficulty.

The urine examination showed: Right, many pus cells and bacilli. Left, occasional pus cells. No organisms. The bladder showed many pus cells and bacilli. Phthalein test—Intravenous injection showed R. U. Ap. time 20 min. excretion after 30 min. was 2%. L. U. Ap. time 3 min. excretion after 30 min. was 25%. Blood chemistry showed: sugar, 79; N. P. N. 34.2; Urea, 15.8; Creatinin, 1.6; Uric-acid, 3.7. The x-ray film revealed a large calculus approximately the size of an egg, completely filling the pelvis of the right kidney. There was a smaller stone completely filling the pelvis of the left kidney. From that examination it was quite apparent that the right kidney would need to be removed. The stone from the left kidney was removed at the first operation and two months later the right kidney together with the stone was removed. Stones are shown in the accompanying illustration.

In operating upon this patient it was felt that the small amount of function in the right kidney could be depended upon in case there was a temporary loss of function while operating on the left side.

In summarizing I believe that there are many patients with bilateral surgical kidney disease that may be operated upon, although at first they appear to be unfavorable cases. In operating upon such patients it is almost always best to operate upon the good kidney first. Spinal anesthesia is particularly useful in certain kidney operations such as the third case reported.

NEPHROPEXY FOR URETERAL KINKS*

BY AUGUSTUS RILEY, M.D.†

THERE have been many different devices and operations described to overcome the so-called "Floating Kidney". The principles of some of these described operations are good and effective, the principles of others are, no doubt, injurious to the kidney. I will not attempt to make any comments, praise or criticisms, on the numerous different techniques found in the review of the literature: because many of the operations, and the results, good or bad, given, were done before the time when a good study of the kidneys and ureters by pyelograms, and ureterograms, and function tests were made or even thought of. Some surgeons even to this day make the statements, not so much in the literature, but before clinics, internes, and medical students, that the operation for "Floating Kidney" is obsolete, having been tried by them and by others and given up as no benefit to the patient. Such statements should not be made, because there is a relief for these patients after a proper pyelographic study, and such a relief can be brought about by nephropexy if properly done without any appreciable damage to the kidney.

Burford reporting forty-eight cases operated upon, says: "General ptosis of the abdominal viscera, kidney and ureteral stones are not contra-indications for the operation. Persistent infection in a ptosed kidney is cured by nephropexy as it keeps the ureter straight so that it can drain the kidney pelvis. Nephropexy is the only relief for the majority of ptosed kidneys causing ureteral kinks."

Mathé after an extensive review of the literature leads us to believe that surgical suspension or nephropexy has fallen in disrepute; not on account of its insufficiency, but because it has been performed when not indicated, the technique has been faulty, or there has been failure to realize the necessity of exposing the ureter and relieving any pathological condition present, which unrelieved will defeat the purpose of the operation.

Clark lays stress on pyelograms and ureterograms to determine the degree of dilatation of the kidney pelvis and the angulation of the ureter. He used the semi-sitting position to get the degree of mobility of the kidney. This position will also show the increased kinking or angulation of the ureter. He says, "No diagnosis should rest solely upon palpation of an unduly movable kidney, unless confirmed by pyelograms to find the part played in giving the symptoms by stricture or kink of ureters." He says, "We have settled upon three cardinal indications for possible surgical intervention in movable or floating kidneys: they are, fixed pain in the renal area, Dietel's crises, and marked vesicle distress referred from the renal area. Even these symptoms, however clear, must be supplemented and confirmed by a rigid clinical investigation, which should include a cystoscopic examination, catheterization of the kidney on the affected side, and in the majority of cases an injection and X-ray examination to ascertain the degree of dilatation of the renal pelvis; any apparent surgical indication not confirmed by these latter diagnostic measures, is in our opinion open to criticism." I fully agree with Clark, but would say that *all kidneys* should be injected and pyelographed before any surgical intervention.

Etiology: The cause of movable kidneys with kinked ureters giving symptoms to a given patient is not so much concern as the relief of the symptoms and its resulting effects. It is found more common in women than in men and is more often found on the right than the left side. It is found in fat and muscular people as well as

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†For record and address of author see "This Week's Issue," page 957.

in thin people. Occasionally kinked ureters are found on both sides in the same patient. I have two such cases, one a woman, the other a man. It is found in early adult life as well as in later years. The mode of dress in women was thought to play an important rôle in producing these conditions, but they are found today, when we feel sure that the mode of dress cannot play any part in causing this condition and giving the symptoms for which the patient seeks relief.

Polak believes that trauma, heavy lifting, rapid repeated pregnancies and shallow renal fossae are the common causes found in women.

Griffith made an extensive review of the literature on the "Etiology of Movable Kidneys," and did some original investigation as to what part conditions found in women played in bringing about the so-called movable kidney. His conclusions were, pregnancies, chronic constipation, severe injuries and gynecological conditions, may in a very small percentage of cases cause nephroptosis, and that the peculiar body shape is not sine qua non: but in the majority of cases they are simply accessories and the true underlying cause, more important than all these factors combined, is the typical body shape.

Congenital anomalies must receive their proper consideration in cases of abnormal mobility of the kidney. The normal mobility of the kidney in women is given by Kelley as 1.5 to 5 c.m. and about half this in men. Young believes that when these limits are exceeded there is an accompanying laxity of the perirenal fascia. The renal vessels become elongated in long standing cases, while the ureter has to adopt itself to the movements of the kidney. The ureter may become fixed at some point by a band of adhesions or an aberrant artery, causing a kink.

Within the past three years I have done nephropexy for kinking ureters on thirty-five cases, twenty-six women and nine men, youngest a girl, fifteen years old, oldest a man, sixty years old. Two cases had two operations, nephropexy on both kidneys, at different times. They felt so much relieved from the first operation that they readily submitted to the second operation for the relief of the pain in the other kidney.

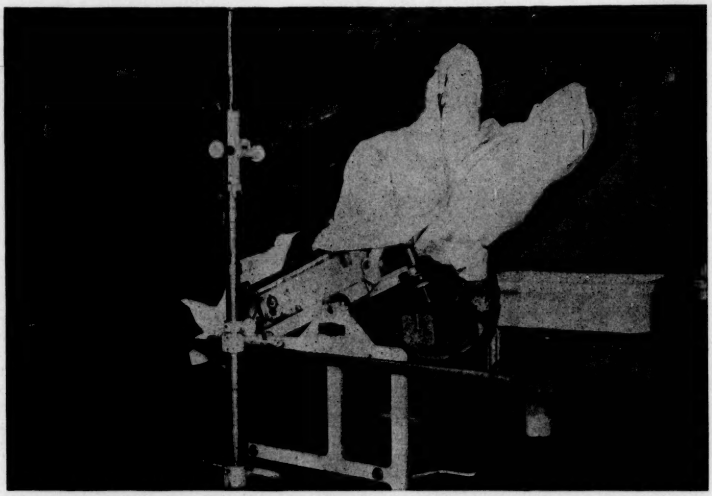


FIG. I. Position of table and patient (Trendelenburg) at the time kidney is injected for plate No. 1.

One case had only one kidney, the other kidney having been removed about ten years previously, the pathology of which she does not know. Four cases had demonstrable pulsating aberrant vessels, causing the kink of the ureter. One case showed at the time of operation that the kink was due to a mass distorting the ureter at a definite point. Pathological examination of a section of the mass proved to be retroperitoneal sarcoma. Twenty-six of the thirty-five cases showed a definite point of fixation caused by more or less dense bands of adhesions at the point where the ureter kinked, demonstrated at the time of operation.

All of these cases had had attacks of severe pain in the kidney region. Some had pain radiating to the bladder or genitals. Two cases had been operated on for chronic appendicitis

without relief. Some of the cases had had other examinations for symptoms described as gastro-intestinal and had been found negative.

All cases have been relieved of the symptoms for which they sought relief. Sixteen of thirty-five cases have been pyelographed six to twelve months after operation and the kidneys found high up in the position they were placed at the time of nephropexy. All cases pyelographed after operation are still free of the kink of the ureter.

Technique of Pyelographic Study: The patient should have bowels cleaned thoroughly of all faecal contents and gas. This we attempt to do by having the patient take about 4 to 5 ounces of Russian Oil the late afternoon before the day the study is made. Faecal matter and gas in the bowel will often give poor plates for study.

The X-ray table used should be one that will place the patient first in the Trendelenburg position and then in the semi-sitting position. The first X-ray plate is taken with both catheters in place and the patient in normal position, that is, flat. This first plate is used as a key plate to detect any calculi in the ureters, kidneys, or kidney pelves or calcified glands that have some relative position near the urinary organs. The patient is tipped into the Trendelenburg position (Fig. 1) and the first injection is made, using a 20 c.c. syringe. With the patient in this position gravity aids the filling, and very little force is necessary to make the injection. The exposure is made as soon as the patient feels the least pain in the kidney being injected. This gives us plate No. 1. The table is then immediately tipped into the semi-sitting position (Fig. II) for plate No. 2. The catheter is pulled down so that the tip will be about the level of the brim of the bony pelvis, and a small amount of the pyelographic medium is injected to make sure of a good ureterogram. Before plate No. 2 is taken the patient is instructed to take three or four deep inspirations to get the greatest mobility of the kidney, and the exposure is made as the patient holds the last deep inspiration. While these X-ray plates are being developed, which takes five to eight minutes, the kidney thus taken is allowed to drain, so as to relieve the patient of any distress before any injection of the other kidney is attempted. The same procedure is gone through in making plates of the other kidney. The plates of the second kidney taken five to eight minutes after the first kidney will give much information as to how completely the first kidney has emptied itself. If the plates of the last kidney taken show a hydronephrosis or kinking of the ureter a final plate is taken five to eight minutes after the injection. The final plate, taken five to eight minutes after the last plate of the second kidney injected, and ten to fifteen minutes after the injection of the first kidney, is sometimes very important and will give valuable information as to difference in the ability of the kidneys to drain.

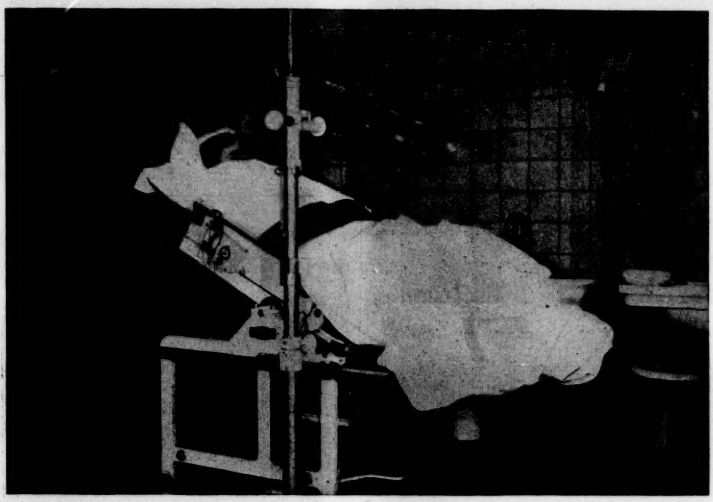


FIG. II. Position of table and patient (semi-sitting) for plate No. 2 (ureteral catheter is pulled down for ureterogram).

Sodium Iodid 12% solution is used. The solution should always be heated to more than body temperature. By so doing there is less

chance of causing pain in the kidney injected. If there is pain which is not relieved by drainage and the plate shows there is a kink of the ureter, the patient should be placed in the Trendelenburg position and the catheter of that ureter introduced higher up in the ureter to establish drainage.

CASE I. (a.) Pyelogram of left kidney before nephropexy.

CASE I. (b.) Pyelogram of same kidney twelve months after nephropexy.

I wish here to report a few of the cases from this group of thirty-five, showing pyelograms before operation and pyelograms made six to twelve months after operation.

CASE I. Female, Nurse, thirty-five years old. For several years at different times she has had attacks of severe pain in region of left kidney, radiating down towards bladder and genitals. Attacks of pain were so severe she would have to give up her work. She had to have morphine for relief, became an opium addict and was reported to the narcotic officers for forging prescriptions for opium. She had a right nephrectomy some ten or twelve years before. The reason for the nephrectomy she does not know. The time her first pyelogram, Case I (a), was taken she had just got over an attack of severe renal colic, when she had complete anuria for fifty-six hours.

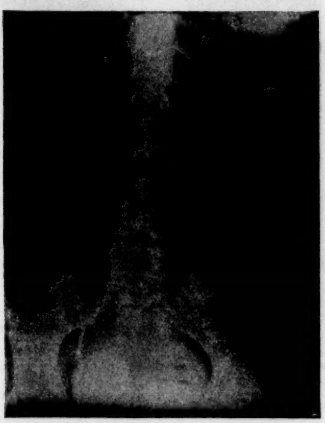
CASE II. (a.) Pyelogram shows a looping-kink of left ureter and some hydronephrosis.

CASE II. (b.) Pyelogram of same kidney six months after nephropexy. Ureter straight, no hydronephrosis.

She refused operation at first and it was more than a year later when she consented to be operated.

The following pyelogram, Case I (b), was taken one year after operation. She has been able to do

CASE III. (a.) Pyelogram shows distinct kinking of ureter, due to aberrant vessel.



CASE III. (b.) Pyelogram taken six months after nephropexy.

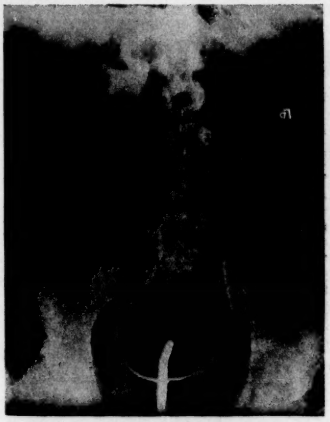
her work, get along without morphine, and has gained twenty-five pounds in weight.

This case now has some pain at times which is, no doubt, due to a pyelitis. Pelvic lavage gives her complete relief for the time and for some time afterwards.

CASE II. A robust young man, twenty-three years of age, came to the clinic complaining of attacks of severe pain in the region of the left kidney, radiating down to the genitals. He thought his pain was due to his work, which called for his lifting heavy articles.

Pyelograms, Case II (a), were made and showed

CASE IV. (a.) Pyelogram of right kidney with a kink of ureter.

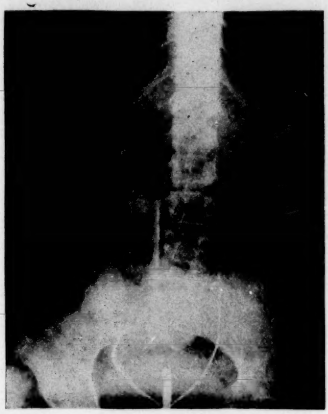


CASE IV. (b.) Pyelogram taken twelve months after nephropexy.

a distinct looping-kink. There is some hydronephrosis with blunting of the calices.

The accompanying pyelogram, Case II (b), was taken six months after nephropexy was done. It shows a normal appearing kidney pelvis. He reports he has been entirely free of his former pain and he has been doing his former work. There is no hydronephrosis, and good cupping of the calices. The ureter is straight, but there is some narrowing of the ureter at the point where the kink was.

CASE III. A young woman, graduate nurse, who had an appendectomy done about seven years before she came to me for pain in the right side of abdomen, pain was similar to that she had before she was operated, but lately the attacks of pain were more frequent and severe. She was unable to do her work because of severity of the pain. Pain was worse when she was standing. Sometimes she was taken with severe pain when asleep if she happened to be lying on her back. Some frequency of urination after an attack of pain.

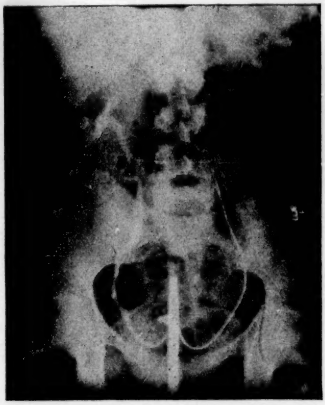


CASE V. (a.) Pyelogram of right kidney showing kink of ureter, hydronephrosis, and blunting of calices.

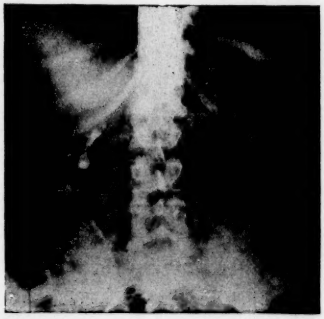
CASE V. (b.) Pyelogram taken nine months after nephropexy. Notice the striking results obtained comparing (a) and (b) pyelogram.

Pyelograms were made. Pyelograms of the right side, Case III (a), showed a distinct kinking of the ureter. There is some hydronephrosis with blunting of the calices. Nephropexy was done, and an aberrant vessel was found at the point where the ureter was kinked. After freeing of the kidney and ureter, the ureter was found to sag over the aberrant vessel. The vessel was ligated and cut.

The accompanying pyelogram, Case III (b), made six months after operation shows a striking result. The kidney pelvis appears normal in outline. The calices have regained a normal appearance and the ureter is straight. This patient has been relieved completely of her pain and is able to perform her duties as a nurse without any loss of time as heretofore.



CASE VI. (a.) Pyelogram shows kinking of ureter.



CASE VI. (b.) Pyelogram taken six months after nephropexy.

CASE IV. Young married woman who was seen in consultation for severe pain in region of right kidney. She gave also a history of severe pain at times in the left kidney region. After attacks of pain in either side she was unable to attend to her duties for some days afterwards.

Pyelograms were made of both kidneys. The right kidney, Case IV (a), showed a hydronephrosis and a kinking of the ureter. The left ureter was straight, but showed a much constricted area about the level of the third lumbar vertebra.

Nephropexy was done and she was relieved completely of her right side pain, but had attacks of pain in the left kidney region. Dilating the left ureter gives her complete relief.

The accompanying pyelogram, Case IV (b), shows the condition of the right side twelve months after

CASE VII. (a.) Pyelogram showing kidney rotated more than forty-five degrees. Ureter in its upper position about the mid line of spine. Some hydronephrosis. Rotation caused by aberrant vessels branching from lower aorta and entering lower pole of kidney.

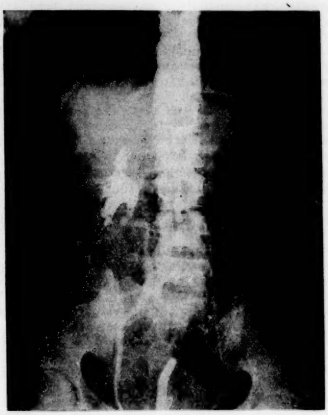
CASE VII. (b.) Pyelogram taken eight months after nephropexy.

nephropexy. The ureter is straight, and the hydronephrosis shows some improvement.

CASE V. Young married woman who gives a history of attacks of severe pain in right back. For past three weeks she has had more or less constant pain with intervals of sharp attacks. Has some frequency of urination.

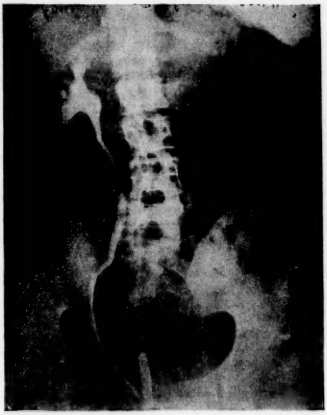
Examination shows a tender mass on the right side; which is movable with respiration and is probably the kidney.

Pyelogram, Case V (a), shows right ureter kinked near its junction with kidney pelvis. There is a right hydronephrosis.



CASE VIII. (a.) Pyelogram of right kidney shows kink of ureter with some hydronephrosis.

The accompanying pyelograms, Case V (b), was made nine months after operation. The ureter is straight, and there is no apparent hydronephrosis.



CASE VIII. (b.) Pyelogram taken thirteen months after nephropexy.

The kidney now empties itself completely in five minutes.

She has had no pain as formerly and feels well.

CASE VI. An Italian woman, who had been operated on a short time before for a right femoral hernia. While doing her housework she was seized

with sudden pain in the region of the right kidney. She was seen by her local physician and sent to the hospital with the diagnosis, "Perinephritic Abscess." When first seen at the hospital she was having severe pain in the region of the right kidney, with much tenderness and spasm in the region. Temperature was normal; a catheterized bladder specimen of urine was negative. White blood count was normal.

A tentative diagnosis of "Blocked Ureter" was made. Heaters were applied to the costo-vertebral area. Morphine was given for pain and the foot of her bed was elevated.

Her pain subsided and two days after entering the hospital pyelograms were made. The pyelogram, Case VI (a), here shows a definite kinking of the ureter. There is no hydronephrosis.

A nephropexy was done and the following pyelogram, Case VI (b), shows the results six months after operation. The ureter is straight and the pelvis appears normal. She has been free of pain and able to do her routine work.

CASE VII. A traveling salesman, fifty-four years old, has had attacks of severe pain in right costo-vertebral angle, extending down to genitals. There was urinary frequency during and after an attack of pain. He was sent to the hospital with the diagnosis of "Renal Stone". Pyelogram, Case VII (a), was made and showed the right kidney rotated more than forty-five degrees, so that the ureter in its upper portion is near the middle line of the spine and a kinking of the ureter at its junction with the kidney pelvis. There is a hydronephrosis with some blunting of the calices. The kidney is low.

Nephropexy was done. When freeing the kidney a large aberrant vessel was found entering the lower pole of the kidney and apparently from its direction it branched off from the aorta low down. The aberrant vessel was large enough to demonstrate its pulsations. It was impossible to bring the kidney into its normal position before ligating the vessel. This vessel was tied and ligated. This caused a blanched area about the size of a ten cent piece on the surface of the lower pole of the kidney. The ureter was freed and the kidney placed in its newly made bed.

The accompanying pyelogram, Case VII (b), was made about eight months after nephropexy. The patient has been relieved entirely of his pain and able to do his work without any interruptions.

CASE VIII. A young married woman, who for some time has had attacks of severe pain in region of right kidney, extending down to bladder. She thinks the pain was caused by her lifting her child. There is some frequency of urination after an attack of pain.

Examination showed some tenderness in right costo-vertebral angle.

Pyelogram, Case VIII (a), showed a marked kinking of the ureter and some early hydronephrosis.

Nephropexy was done and the accompanying pyelogram, Case VIII (b), made thirteen months after operation shows a normal appearing pelvis and straight ureter. The kidney pelvis empties itself completely in five minutes.

Operation: Nephropexy should be accomplished with as little damage as possible to the kidney itself. I can't help but feel that a silk suture or any slowly absorbed or non-absorbable material introduced through the cortex of the kidney will in time cause damage to that kidney. I feel also that the least possible drag on the kidney should be avoided. Therefore I think we should avoid suspending the kidney by placing a suture of any kind through the cortex of the kidney.

The true capsule of most kidneys is too friable to bear any weight of any consequence and is not flexible enough to allow any excursion of the kidney with the respiratory movements. Therefore with the above possibilities in mind I have attempted to avoid any possible damage to the kidney and not to rely wholly on the friable true capsule to hold the kidney in its new bed where it has been placed: but have utilized the fatty capsule in such a way as to hold the kidney in its newly placed position.

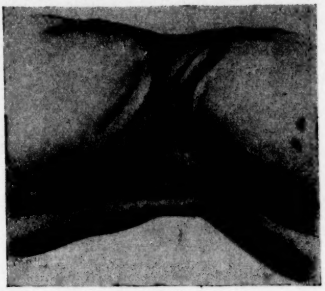


FIG. III. Position of patient on operating table. (A) Incision.

The patient is placed on the table on the side (Fig. III) with sandbags underneath, to bend the patient as much as possible, giving plenty of space between the crest of the ilium and costal margin. The so-called "hockey-stick" incision is used. The incision extends from the lower border of the twelfth rib at the outer edge of the lumbar muscle down to near the crest of the ilium. All underlying muscles are cut across.

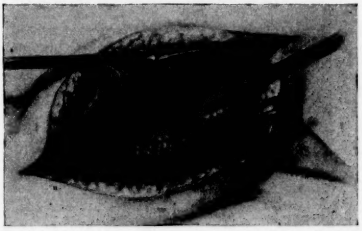


FIG. IV. Muscle cut across. Fatty capsule (B) in view and incised so as to be dissected downward and later utilized for the main support of kidney.

The perirenal area is exposed. The fatty capsule (Fig. IV) is incised at the upper part of the perirenal area so that the capsule can be dissected downward and later utilized to make a rest or hammock to support the kidney. The kidney is freed of adhesions. The ureter is

freed down below the point of kinking as demonstrated by the X-ray plates. Aberrant vessels are looked for and the relation, if any, that they may bear towards producing the kink of the ureter is demonstrated.

A new bed is made above the costal margin against the rib-cage up to the diaphragm. The freed kidney is placed into its newly made bed: at the same time noticing the straightening of

FIG. V. Kidney delivered into wound. True capsule split along convex surface of kidney to about one inch of lower pole. Flaps (C, C) dissected down to hilum of kidney.

the kinked ureter and the amount of tension thus placed on the straightened ureter.

The true capsule of the kidney is split from the upper pole of kidney along the convex surface down to one inch of the lower pole of kidney(Fig. V). The flaps of the capsule still attached to the lower pole of kidney are dissected down on both sides to near the hilum of the kidney.

The kidney now is pushed up within its newly

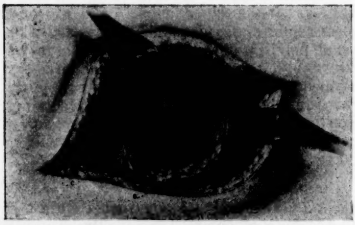


FIG. VI. Flaps (C) of true capsule sutures to Quadratus Lumborium muscle. (B) Fatty capsule.

made bed and held by an assistant while the flaps of the true capsule are sutured with a No. 1 plain catgut to the under surface of the cut quadratus lumborium muscle (Fig. VI). This brings the raw surface along the convex surface of the kidney in contact with the area of the newly made kidney bed.

The next and final step, which I feel is most important, is the utilization of the fatty capsule to support the newly placed kidney. The true capsule I feel is too friable to withstand the kidney weight and more especially to resist the respiratory tug on the kidney. The fatty capsule is tucked in under the lower pole of the kidney and sutured to the under and cut surface of the quadratus lumborium muscle along the same line of sutures made with the true capsule and quadratus lumborium muscle. This makes a support for the kidney and holds it up, and at the same time holds it back against the surface of the rib-cage (Fig. VII). With gathering sutures the excess of the fatty capsule is caught up in such a way so as to obliterate the space in which the kidney was, but still allow some excursion with the respiratory movements.

A cigarette drain is placed in the depths of the wound and brought out at the upper end of the skin incision. This is done to take care of any accumulation of blood serum. The drain is removed in forty-eight hours.

The severed muscle edges are carefully approximated with No. 2 interrupted chromic cat-

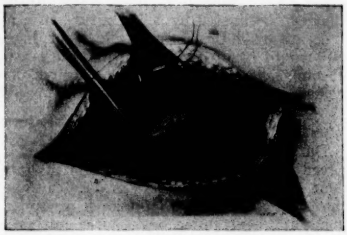


FIG. VII. Fatty capsule (B) sutured to quadratus lumborium muscle along line of sutures of true capsule and quadratus lumborium muscle.

gut sutures. Skin wound closed with interrupted silk worm gut sutures.

Apparently there is very little or no shock caused by the operation.

The patients are kept in bed for two weeks and then allowed to be up and around and given permission to perform their regular duties when they feel strong enough to do so. No support of any kind is worn.

Conclusions: Nephropexy for floating or mobile kidneys with kinking ureters will give relief of pain, establish proper drainage, and relieve early hydronephrosis.

Nephropexy should not be done for symptoms and clinical findings alone, but symptoms and clinical findings combined with the confirmatory findings of pyelograms and ureterograms, taken in both Trendelenburg and semi-sitting positions.

A recurrent or still existing pyelitis or stricture or narrowing of ureter at the point of the previous kink are the most common causes of pain after nephropexy.

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Discussion of Dr. Deming's Paper

Dr. William C. Quinby, Boston: The importance of this work is self-evident. Anybody who must enter the kidney to get out a stone must know the best way of approach and of subsequent suture. Experimental work on this subject is important. I would like to ask Dr. Deming how he has fared in the human type of kidney, for in man one has not all the factors of safety that one has in the laboratory dog; as the kidney may be damaged before operation by disease. Sometimes under these circumstances there is very little bleeding anyway; and under other circumstances one finds a definite bleeder that can be ligated by a fine suture on a needle. I do not make these remarks in a carping spirit but would like to emphasize that we must have at our command all types of sutures, and be versed in all the various operative procedures.

Discussion of Dr. Hepburn's Paper

Dr. Clyde L. Deming, New Haven: I have never resected a kidney in a human but have done several single kidneys in dogs carrying out the idea of suturing that I have presented. It is not a difficult procedure. After cutting across the kidney with a sharp knife, the ends of the kidney can be brought together with this type of suture easily. The corrosion model of such an operated kidney shows little less than half a kidney, indicating that there is little blood vessel injury.

I think Dr. Hepburn's idea of separation is an excellent one if it can be done as he describes.

Question: How did it function afterwards?

Dr. Clyde L. Deming: The function of an experimental heminephrectomized kidney is about 40% of normal when the opposite kidney remains, Bilateral heminephrectomized kidneys each function about 60% normal.

Dr. George G. Smith, Boston: I remember one case that had a much dilated upper calyx which had made a local hydronephrosis and, of course, resection under such circumstances is a simple matter. I have never done a true resection of the kidney in the sense that Dr. Hepburn describes.

Dr. William C. Quinby, Boston: The success of the operation of heminephrectomy depends in each instance on the reason for its performance. Should there be present in the kidney an infection such as tuberculosis, even if this be definitely localized so far as appearance to the eye is concerned, removal of half such a kidney would be very dangerous, because you have no assurance that the other remaining half of the organ may not also be somewhat involved by tuberculosis.

Dr. Hepburn's case is very interesting, and of course the patient was very fortunate in happening to have a kidney so well demarcated into an upper and lower half. This is rather an uncommon anomaly.

Question: Has he now sterile urine?

Dr. Thomas N. Hepburn: Yes.

Dr. Quinby: He had a great deal of infection?

Dr. Hepburn: Surely.

Dr. Quinby: It only goes to show what the extent of the reparative processes of the kidney may be.

Dr. Roger C. Graves, Boston: I would like to congratulate Dr. Hepburn on the conduct of his case because it was a difficult problem and he obtained a splendid result.

The only experience I have had which approaches it occurred in a case which I saw recently with Dr. Weiner of Brockton. The patient was an individual with congenital absence of the left kidney and a large renal tumor on his right side. Because of the extent of the mass and the pressure symptoms which were resulting from it, it seemed imperative to advise operation. We found a very large solitary cyst —the largest one I have ever seen—arising from the broad anterior surface of the single right kidney. It contained about 1,500 c.c. of clear serous fluid and had no communication with the renal pelvis. The cyst was resected in its entirety, and the patient made a satisfactory recovery though the convalescence was tardy because of a cardiac complication. He is now in excellent general health. That is my closest experience to Dr. Hepburn's; the case will be reported in detail, later.

Dr. Thomas N. Hepburn, Hartford (closing): I have only to say that I didn't cover all the cases of partial kidney resection, but the majority of resections have been for single solitary cyst. Caulk has done a partial resection for dilatation of a calyx. A number of resections for tuberculosis have been done but none of them were satisfactory. I have resected solitary carbuncles in two cases. I didn't diagnose them beforehand, but operated for perinephritic abscess. I took a wedge including the carbuncle out of those kidneys and sewed them up tight. They have done well. I think solitary cyst and dilated calyx with stone will represent the pathology in the majority of cases calling for partial resection.

DISCUSSION OF DR. NEUSWANGER'S PAPER

DR. WILLIAM C. QUINBY, Boston: The question as to which side to operate upon first, having decided to operate at all, is always an interesting one. What led you to operate on the better side first?

DR. C. H. NEUSWANGER: I had a case a year before in which I did the other side first and did not get along well. And this patient's right kidney wasn't very good but still it had 2% of function, and I have an impression that doing an operation such as this patient required on the kidney does a good deal of trauma and throws the kidney out because there is a good deal of trauma in taking these stones out. and I think that 2% of function in the bad kidney may have pulled the woman through.

Another thing I do not understand is why she was so sick at the first operation and not at the second.

QUESTION: What was the function?

DR. NEUSWANGER: At the second operation it was around 30% though at the first it was two.

DR. HOWARD M. CLUTE, Boston: Is it not your custom to operate on the good kidney first?

DR. QUINBY: One must decide each case for itself. In some instances where treatment of the more damaged side must be by nephrectomy, this operation should precede relief of the less damaged side. Other cases will frequently be found, however, in which it is best to make some relatively minor operation such as pyelotomy or nephrotomy, on the better kidney before interference on the more damaged side. I do not feel that we are able to lay down any hard and fast rule in this matter.

DISCUSSION OF DR. RILEY'S PAPER

DR. CLYDE L. DEMING, New Haven: How many cases were completely relieved of pain?

DR. WILLIAM C. QUINBY, Boston: I would like to call your attention to the fact that in all these cases Dr. Riley got the kink out of the ureter, as judged by the postoperative pyelogram. In some instances one finds that even after a nephropexy has been done the ureter is not straight. The incidence of abnormality on the part of the blood vessels is important. I have been interested in this subject for a number of years, and have seen a few cases similar to that described by Dr. Riley in which an aberrant vessel arises from the aorta below the kidney, being attached to the kidney in a way so as to hold it down. A kidney held low by a vascular anomaly is definitely relieved by section of the vessel.

DR. ERIC STONE, Providence: I would like to ask about the occurrence of unusual pain in the immediate and late convalescence of the patient because sometimes one is disturbed after nephropexy by the pain the patient has and for some months after.

DR. QUINBY: Pain of what type?

DR. STONE: Pain in the region of the kidney not of the type they had before operation.

DR. QUINBY: Of course, the question of the advisability of operation for movable kidney is an omnipresent one and like all other things in medicine it is subject to changing opinion. No doubt, a number of years ago, there were too many nephropexies done, often on little or no indication whatever; and the conservative ones among us felt that it was an operation that was hardly ever justifiable. This point of view was too extreme, and I am perfectly sure that for sufficient cause, nephropexy is a proper operation and when properly done, followed by marked relief.

The indication must be clearly outlined and defined, and it has been possible to do this because of increasing detail in our ability to study patients. We study them now better than we did twenty years ago, and especially do we examine patients in whom a question of the advisability of nephrorrhaphy is raised, by the method of pyeloscopy. If the injected renal pelvis empties within normal time limits it is highly probable that fixation of that kidney will do no good. So I think the pendulum as regards advisability of operation has reached the point where nephropexy is an operation which should be done for adequate cause.

DR. ROGER C. GRAVES, Boston: I want to congratulate Dr. Riley on his paper and his work. I was much interested in his case in which the patient found it advantageous to lie on the abdomen and not on the back for the relief of pain. It recalled an amusing experience in the laboratory some years ago when I encountered in a cat an unusual anomaly in the relationship between the right ureter and the vena cava. The ureter instead of being lateral to the cava, crossed beneath it a short distance below the kidney and then extended downward along its medial side to the brim of the pelvis. There it crossed the cava again, this time in front, and then continued in a normal course to the bladder. There was no apparent interference with drainage in the ureter, and the kidney of the cat seemed to be normal in all its gross characteristics.

In looking up the literature to see if any such condition had ever been recorded in the human, I discovered in the German reports four similar cases, in all of whom there had been, as a result of the anomaly, a definite hydronephrosis. I went to Dr. Lewis for light on the embryological features of the problem. He was very helpful

and the whole matter was discussed in detail in the *Journal of Urology* (July 1922). I told Dr. Lewis that the records described a hydronephrosis in the humans who presented this anomalous relationship between ureter and cava, while my cat showed no apparent ill effects. He replied that the humans had suffered from the lack of a quadruped position and referred me to a paper which he had written while in college, on the unfortunate upright position of man.

DR. AUGUSTUS RILEY, Boston (closing): In answer to Dr. Deming, this first case has had attacks of pain but not so severe as formerly and unquestionably pain is due to an existing pyelitis. Another case had pain after nephropexy, pyelograms showed the kidney well up in position and no delay in drainage time. On examination it was found that the pain was not similar to the pain before nephropexy but more superficial so it was concluded that the pain was due to a nerve caught in the operative scar. Dissecting out the scar gave her complete relief. The husky young man with a kink of the left ureter had a good deal of pain after operation, apparently a nerve was caught in one of the sutures and he had pain for several days but was free of pain before he left the hospital.

In answer to Dr. Colby:—Not all of these cases have hydronephrosis, some show no hydronephrosis. The question of hydronephrosis in some of these cases has been discussed several times with Dr. P. F. Butler, Roentgenologist-in-Chief, Boston City Hospital. I feel that these cases do not get the dilatation we might expect because when an attack of pain comes on, it is so severe that the patients have to go to bed and they have to have morphine, and they soon learn that getting into certain positions gives them relief from pain. One case had his attacks of severe pain in the mornings shortly after getting out of bed, especially if he jumped up quickly. In other words, very few of these cases have much, if any, dilatation of the kidney pelvis, because the pain they have is so acute that they either have morphine or find some position of the body to relieve the kinked ureter.

Dr. Graves's laboratory case of the cat was interesting. There was a case of nephropexy done at the Boston City Hospital a short time ago in which at operation a large vein, probably the main renal vein, was found to run along the deep muscles of the back and empty into the vena cava low down.

DEDICATION OF NEW UNITS OF CAMBRIDGE HOSPITAL

Three additional units of the Cambridge Hospital, which overlooks the Charles River at Mt. Auburn street, Cambridge, were dedicated October 29 with exercises held in the auditorium of the new Nurses' Home, attended by 400 prominent men and women.

Yesterday marked the 43d anniversary of the opening of the institution. The three new buildings afford accommodations for 316 patients.

Prof. Francis G. Peabody presided at the exercises. The hospital nurses were ushers. Judge Robert Walcott, a member of the board of trustees, represented his father, Dr. Henry P. Walcott, who was unable to attend. Judge Walcott read a paper prepared by his father relating many interesting reminiscences of the romantic history of the hospital. Dr. Walcott was the first president of the institution and is still a member of its medical and surgical staff.

Rev. Raymond Calkins offered prayer and Mayor Quinn of Cambridge spoke, praising the fine-spirited men and women who made possible the erection of the new buildings.

Prof. Byron S. Hurlburt of Harvard University made the speech of presentation. In the address of acceptance, George A. Giles, treasurer of the institution, expressed the gratitude of the trustees, and predicted the appreciation of the community.

"By these buildings," continued Mr. Giles, "you have erected a monument to the spirit of unselfishness which can crumble only when it no longer becomes necessary for you to minister among each other. Today you have expressed your approval of those who builded before you and have added your testimony of devotion to this great cause."

Following a description of the three new units in minute detail, by Dr. James W. Seaver, orthopedic surgeon, a tour of inspection was conducted.

Officers of the hospital present at dedicatory ceremonies included Dr. William D. Swan, president; James L. Paine, vice-president; Edward Ingraham, secretary; George A. Giles, treasurer; George Howland Cox, William H. Pear, Jeremiah Smith, Jr., Robert Walcott, Walter F. Earle, James R. Jewett, Edmund S. Kelley, Thomas Hadley, John T. G. Nichols, Edward Ingraham, Robert W. Knowles and George L. Dow, trustees.

The new Morrill Wyman House is a large five-story structure facing south on the Parkway overlooking the Charles River and Soldiers Field. The four large sun porches command an excellent view of the surrounding countryside. This structure has accommodations for 59 adults. There are five nurseries with facilities for the care of 48 babies. An elevator runs to the roof, where a roof garden will eventually be established.

The new four-story Woolson Building is devoted to the care of children. Facilities provide for about 85 patients. This structure also has spacious sun porches with a view of the winding Charles. A large solarium and a series of special rooms provide for sunlight treatment.

The new Nurses' Home is connected with the original home and located to the south of it. It is large and attractive with every modern equipment. It will eventually house 110 nurses.—*Boston Globe.*

THE NEW HAMPSHIRE MEDICAL SOCIETY

OEDEMA*

BY ROGER I. LEE, M.D.†

IN the last century most medical writers felt confident that they understood oedema. Oedema was the fluid part of the blood in the tissues. In heart disease the failing heart could not cope with the volume of blood and bring it to the kidneys, hence stagnation occurred in dependent parts of the body and dependent oedema. In kidney disease there was some blocking of the excretion of urine, hence a generalized forcing of fluid into the tissues. Thus there was the important clinical differential sign of oedema of the face in nephritis. In those days the oedema of cirrhosis was obviously obstructive. Even more obviously obstructive was the oedema of phlebitis, or of pressure of growths. In other words the explanation of oedema was on a mechanical basis. In those days despite this mechanical theory of the production of oedema, energetic measures were undertaken to rid the body of oedema through the kidneys or diuresis. Of course digitalis and the various heart tonics were employed as diuretics. The familiar observation of diuretic effect of coffee led to the use of caffeine and other members of the xanthin group as diuretics. Purgation was in vogue. Calomel was often used in large doses. There were occasional observations of the profound diuretic effect of calomel.

Roughly beginning with the present century more accurate chemical observations were made on the urine and blood of patients. Animal experimentation was utilized to study various abnormal physiologic phenomena. It became evident that oedema was by no means a simple process in every instance. There was a tendency to view oedema from a chemical basis in large part. It was hoped that the vast studies of blood chemistry might find the solution. Progress indeed was made but the solution was not obtained nor is it yet.

We now can carefully and cautiously define oedema as an excessive or pathologic accumulation of lymph of normal or altered composition within the cells, intercellular spaces or serous cavities of the body. (Wells, McLean.) That really means little more than a mild acceptance of the present day view of the chemical nature of the body. Valuable as the study of blood chemistry has been, we now recognize that in many ways the blood is the intermediary between the cell and the urine. From the urine we can construct a rough picture of the bodily or cellular metabolism. From the blood which is nearer the cell, our picture is perhaps clearer and more detailed but still rough and far from a complete and accurate picture of the cellular metabolism.

In the production of oedema we recognize now many factors, which act differently in different cases. Some of these factors are mechanical, others chemical in the usual sense, others physical in the usual sense. Even in the oedema of heart disease where the initial physiologic disturbance is mechanical and dynamic, the precise mechanism of the production of oedema, of its continuation, and of its disappearance is by no means clear.

In the oedema of nephritis we are even more in the dark. There is certainly a tendency to attribute more emphasis to extra renal factors. For example there is a somewhat bewildering condition, usually called nephrosis. The salient features of this condition are marked oedema, marked albuminuria, without red cells in the sediment. The blood pressure is not elevated. There is no retention of nitrogenous substances. The blood shows a reversal of the ordinary ratio of serum albumin and serum globulin. Occasionally one of these cases dies of an intercurrent infection usually and even at autopsy there is a dispute as to the existence of kidney pathology in the accepted sense. To be sure with that picture the burden of proof may be to show that the kidneys are not damaged in some functional fashion. Nevertheless the association of marked anasarca in some gastric cases, in anemia, in starvation, and in other conditions, likewise influence the tendency to emphasize extrarenal and extracardiac conditions in the production of oedema.

It is perhaps natural that in the course of this rather bewildering turn of events, attention to attempts to rid the body of oedema has been scanty. Indeed even the diuretics already available were almost neglected. In partial explanation of this attitude it should be said, first, that a better understanding of so-called cardio-renal disease with oedema, usually showed that the decompensated heart was the underlying cause of the oedema. In the last few years digitalis therapy has been made vastly more effective. Therefore attention is directed sometimes too exclusively to digitalis therapy. (Perhaps some of our heart specialists would feel that diuretics were out of their sphere. Another wicked suggestion is that digitalized patients frequently die suddenly and not after a protracted period of dropsy.) Certainly the cases of massive anasarca are no longer common.

A second cause for the neglect of pharma-

*Read before the Annual Meeting of the New Hampshire Medical Society at Manchester, May 29, 1929.

†For record and address of author see "This Week's Issue," page 957.

ceutical diuretics is, I believe, the elaborate chemical metabolism studies in nephritis. As I have said previously, from the chemical studies everything was expected and much was obtained. Such fundamental concepts as the important relation of sodium chloride and water to nephritis and oedema were established. Brief mention should be made here of the Karell diet, that is, the administration of 200 c.c. (or a glass) of skimmed milk 4 times a day for 4 to 7 days. No other food or water is given. Often this is still for a short period a most satisfactory low salt, low fluid diet and is accompanied by the control of oedema. We probably overemphasized the importance of nitrogen retention in the blood and the desirability of protein restriction in the diet. Nevertheless as digitalis controlled in many instances the oedema of heart disease, so salt and water restriction helped to control the oedema of some renal cases.

Latterly attention is once more turned to the pharmaceutical diuretics and these diuretics are being tested with all of the newer chemical, physiologic and experimental methods. Happily it is being found that there are very valuable diuretic agents, in addition to digitalis and the heart tonics, and the low salt, low water diet. Curiously enough the method of action of these substances is not clearly understood. Perhaps if it were, the nature of oedema itself would be more clearly understood.

It is, I think, very pleasant to note that under modern careful scrutiny some of our older and rather neglected friends of the xanthin group (Caffeine, theobromine, theophyllin compounds) are found to be valuable. It will be noted that in this connection I do not differentiate sharply between cardiac and renal oedema. Although there is still some hesitation in employing a drug known to be a kidney irritant in renal oedema, there seems to be little difference in the action in the type of oedema. And we have already suggested the general identity of oedema. Of the xanthin group, diuretin or theobromine sodium salicylate in daily doses of 60 to 150 grains, theocin, or theophyllin, in daily doses of 15 to 30 grains, euphyllin, a theophyllin derivative, in daily doses of 5 to 15 grains, are all occasionally but not uniformly effective in the order given. They should be given for a few days only at a time. They are not as effective generally as some of the diuretics about to be discussed, but they are nearly always safe. Gastro-intestinal disturbances are not uncommon. I want to repeat again that these drugs should be given after digitalization in heart cases and with appropriate diet in renal cases.

Only within a few years was it found that certain salts, notably ammonium chloride, ammonium nitrate and calcium chloride, would in certain cases produce marked diuresis. To get this effect one of these salts must be given in large doses, 75 to 150 grains a day. The effect of the ammonium salts at least is to create an acidosis, the urea in the blood is increased and there is loss of the alkaline bases of sodium and potassium in the urine. In other words this bugaboo of recent years, namely acidosis, seems to have some connection with a beneficial diuresis. It is certainly bewildering and confusing. However, it is not as simple as a simple acidosis because the administration of acids does not produce a diuresis. Furthermore these substances act rather differently. Urea is a well known diuretic but the ammonium salts which cause an increased urea in the blood are more effective diuretics than urea. Calcium chloride which is also an effective diuretic does not cause an increase in urea and perhaps is best given when nitrogen retention is demonstrated or suspected. In any event, frequently, but not always and not uniformly, in apparently similar cases these salts administered in these large doses of 75 to 150 grains a day will cause a very marked diuresis in oedema associated with heart disease, cirrhosis of the liver and some types of kidney disease, especially the nephroses. The administration may be continued over several weeks. With nitrogen retention one might prefer calcium chloride or the xanthin group.

It has long been known that calomel and other mercurial salts occasionally produce a marked diuresis. In the minds of most of us before the advent of those newer diuretics, calomel, I think, stands prominent as a diuretic with colossal possibilities. To be sure the successful cases were not frequent, but the occasional case with a loss of 20 to 40 pounds with a corresponding diuresis of 48 hours always remains vivid. But calomel 2 grains every 3 hours or 3 grains every 4 hours for one or two days, besides being uncertain, often caused diarrhea or stomatitis.

There are several organic mercuric compounds which are very effective diuretics. They are employed almost exclusively either intravenously or intramuscularly. They are usually designated by their trade names. Their chemical formulae are known, but are usually fearfully long. As far as known their actions are generally similar. The best known is perhaps Novarsurol (the trade name) or Merbaphen. One or 2 c.c. are injected into a vein or deeply into a muscle 2 or 3 times a week until diuresis is finished or it is evident that no diuresis will take place. The mercury is mostly promptly excreted through the kidney. There is no demonstrable change in the acid-base balance in the urine or blood. The diuresis is frequently very large but is perhaps not quite so consistent as with ammonium chloride, ammonium nitrate, and calcium chloride. So far most observers have been rather chary of giving mercurial salts with blood in the urine.

In obstinate cases the combined use of one of the ammonium salts or calcium chloride with injection of an organic mercury compound may be tried with benefit.

At present certain bismuth salts are being

tried as diuretics, because in many ways bismuth resembles mercury.

In such a discussion as this many points of importance are omitted. I do not want to burden you with details of the other substances excreted with the water in the urine. As is well known the urine in diuresis approaches water in its composition, but always there is chlorine and some fixed base. Apparently these substances besides the water vary much with the different diuretics involved. But what has always seemed to me particularly extraordinary was that there are no constant changes in the blood during diuresis. There is one laboratory fact of real value, when it can be made available, and that is that if the plasma chloride is well below the normal threshold there is no diuresis. If that fact is known, chlorides should be of course administered.

I have by no means exhausted all the diuretics. I think I have touched upon the most effective of them. There is involved some principle which we do not fully understand and connecting it with osmosis or surface tension or viscosity does not help at the present time.

While the xanthin group may possibly act directly on the kidney, the other diuretics under discussion would seem to have mostly an extrarenal and extracardiac effect. There are probably profound blood or cellular changes which underlie this diuresis. It will be noted that in the case of what I have called the newer diuretics, very large dosage is employed. Smaller dosage seems ineffective. It is interesting certainly that some of the more effective diuretics are associated with an acidosis. Much less commonly, but occasionally, a diuresis will occur in connection with the large administration of alkalies. Alkalosis is however a great danger and seems more to be avoided than acidosis. I think it must be assumed that underlying these phenomena of diuresis in consequence of the employment of these drugs in large doses, there is a very substantial modification of some sort of the human physiology. If and when diuresis occurs with these drugs, the phenomenon is certainly striking. And yet in apparently similar cases no diuresis will occur. Sometimes subsequently a diuresis may be obtained. It seems an unescapable conclusion that the mechanism of diuresis nearly always exists even in the face of marked oedema. Some alteration mechanical, physical, chemical, or perhaps in osmotic balance, in viscosity or surface tension, suffices to set the mechanism of diuresis in action.

Perhaps we would do well to scrutinize diuresis as it occurs spontaneously in disease We have always before us the diuresis of the diabetic and the intravenous injection of a hypertonic solution of dextrose (500 c.c of 20% solution) is a diuretic. Stimulation of certain nerve centers brings diuresis as does abnormality of certain glands of internal secretion. In some cases the administration of thyroid substance acts as a diuretic. There is therefore a fertile field open for the future.

Discussion

Dr. H. T. French, Hanover: *Mr. President and Members*—I am very much interested in what Dr. Lee had to offer in connection with oedemas. In regard to the limiting of protein in the diet in oedema, occasionally we will find that an increase in the protein will do much better than limiting the protein intake. I have in mind two cases in the hospital this spring. One was a farmer weighing 240 pounds who came in with tremendous oedema of the face, abdomen and extremities. In going over his history he told of having lived on a diet of white bread, potatoes and salt pork for five months. I had in mind some of the work that was done during the war on oedema and put him on a high protein diet with limited salt intake and limited fluids, and after a few days he complained bitterly of being unable to sleep at night because of constant travelling from the bed to the toilet. In twelve days his weight had been reduced about 67 or 68 pounds. I think that is the most striking case of loss of fluid that I have seen.

Then there was a case that came in at the same time that had a similar history; that is, both of these cases were due to a low protein diet or protein free diet for continued periods and perhaps had something to do with the high salt intake of salt pork.

I like in these oedema cases to have a check on the blood chloride, and if we have a high chloride, to limit salt intake and limit the fluids, and I think that we get on the average better results with the limiting of chlorides and the fluids than we do with other methods.

If we find that results are not being obtained in that way, we usually start to use the xanthin group of diuretics and leave the mercurials usually to the end. I like the action of salyrgan better than the action of novarsurol. I think that in the cases with "kidney damage" more damage will occur from the use of novarsurol than with the use of salyrgan.

Dr. Granville E. Hoffses, Manchester: I have been very much interested in the paper so ably presented by Dr. Lee. A few weeks ago when our Secretary called me up and wished me to talk on this subject, in my mental aberration I was foolish enough to accept, not realizing the broad field, and the broadness of that field has increased every day and more so today—it has no limit.

I wish to confine my remarks more particularly to the idea of the development of oedema from heart failure and I wish again to emphasize the definition given by Dr. Lee that oedema is usually a pathological increase of lymph in the intercellular spaces or the serous cavities of the body.

We all know that a failing myocardium is the

basic cause in most cases of oedema, exclusive of these caused by acute nephritis and chronic parenchymatous nephritis.

I believe that the great trouble with our treatment of oedema by digitalis among men in general is that we fail to use sufficiently large doses. I have a case in mind of a farmer who came to me weighing 260 pounds, water-logged, hardly able to breathe and with a weak, enlarged heart. He was a heavy eater. He was put on a Karell diet for twelve days supplemented by an antifat diet, and he dropped 27 pounds in 3 days and now he is able to lie down at night in bed and be comfortable. By the way this man had already been treated with digitalis. He had been given what might be considered adequate doses of digitalis not taking his weight into consideration. But when he was given adequate doses based on his weight his pulse rate dropped from 120 to 70.

I do not feel that it is necessary to stress the xanthin group or novarsurol which have been already touched upon. Some recent studies of Richardson and Plant on the kidneys of rabbits and frogs are interesting in throwing more light on the production of oedema in slight kidney disturbances, and I believe that in the course of five years we shall see through chemical studies and the clinical work that is going to be done a great advance in the treatment of oedema and its cause.

I wish to compliment Dr. Lee for the broad way in which he has covered this subject.

DR. D. E. SULLIVAN, Concord: I thought this a very opportune time to have this subject presented to the members of this Society. I realized the importance of it and the real necessity of knowing about it. It had been called to my attention several times very forcibly in the past few years that the use of the newer diuretics and the treatment of oedema if known by the profession in general, was not being observed. As Dr. Frank Billings would say, it is the man out yonder to whom this message should go with the greatest of joy.

Too often do we see patients needing up-to-date treatment dosed by diuretic tablets, or small amounts of citrate of potash or other remedies which are well-nigh useless in these oedema cases. I have used these large doses of ammonium salts with novarsurol after the patients had been digitalized in cardiac cases, and the results in the first few days were amazing; and I tried to pass that personal observation along in talking to other doctors and in consultation, and if we will bear that in mind, there will not be anything of more benefit than the discussion of this subject by Dr. Lee today.

DR. ROGER I. LEE, Boston (closing): I think what Dr. French said about the diets is extremely timely. As I tried to point out, we have been very much overdoing the low protein diet, and there are definite types of oedema which are associated with a low protein diet and which are very much benefited by a high protein diet; and I am sure that Dr. F. C. Shattuck, if he were now alive, would be delighted because he always maintained that he never saw anything sinful in a good beefsteak, no matter if the patient had nephritis.

I am glad Dr. Hoffses emphasized the cardiac origin of oedema because we always have to bear that in mind.

MISCELLANY

NEW MEMBERS

Dr. Herman Christophe, 875 Elm Street, Manchester.

Dr. Charles L. Harris, Berlin.

Dr. John Joseph Boardman, Hanover.

REINSTATED

Dr. Everny P. Lunderville, Berlin.

THE HILLSBORO COUNTY MEDICAL SOCIETY

The Hillsboro County Medical Society met at Manchester, New Hampshire, October 22, 1929 for the twenty-fifth annual meeting.

Officers elected are Dr. W. A. Thompson, President; Dr. H. E. Thompson, Vice-President; Dr. D. G. Smith, Secretary and Treasurer. Board of Censors: Dr. E. A. Jones, three years; Dr. S. G. Davis, two years; Dr. M. H. Towle, one year. Delegates to the New Hampshire Medical Society: Dr. Pierre Bergeron, two years; Dr. B. G. Moran, two years; Dr. W. P. Grimes, two years; one year, Dr. D. W. Parker and Dr. D. G. Smith; member of the medical defence committee, Dr. D. W. Parker.

Dr. Charles A. Weaver of the State Board of Health delivered an address in which he paid high tribute to the physicians of Hillsboro County and showed that the medical needs of that section are fully met.

NEW HAMPSHIRE TUBERCULOSIS ASSOCIATION

Dr. Paul Wakefield of Boston and Mr. Daniel W. MacLean of Berlin, New Hampshire, were the guest speakers at the Annual Meeting of the New Hampshire Tuberculosis Association which was held on Tuesday, October 8, at 12:30, at the Rice Varick Hotel, Manchester, N. H. Dr. Edward O. Otis, Honorary President of the Massachusetts Tuberculosis League, was also at this meeting.

The New Hampshire Tuberculosis Association is one of the foremost tuberculosis organizations in the country and has received nation-wide recognition for the work that it has accomplished. At present the Association is desirous of securing sufficient money to meet the requirements of one of New Hampshire's philanthropic citizens who has offered to build a Summer Health Camp or a small all-year-round Preventorium just as soon as the New Hampshire Association has an endowment fund sufficiently large to assure some part of the maintenance.

Honorable John G. Winant, President of the Association, presided at the Annual Meeting. Other officers of the organization include: Mrs. James W. Remick, James A. Scully, Dr. Arthur L. Wallace, Vice-Presidents; Honorable Fred H. Brown, Mrs. L. M. French, Rt. Rev. George A. Guertin, Honorary Vice-Presidents; Arthur H. Hale, Treasurer; and Dr. Robert B. Kerr, Executive Secretary.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 15451

SLIGHT DYSPNEA WITH IRREGULAR FEVER

MEDICAL DEPARTMENT

First admission. A girl four and a half years old entered April 18. Her parents were Greek and spoke little English. The chief complaints were cough and heart trouble.

A month before admission the parents noticed that the child had rapid heart and pulsation of the chest wall and in the neck. She also tired easily. They took her to the Out-Patient Department of this hospital for weekly visits. The records show that the chief complaint was abdominal pain recurring three or four times during the past two years and causing her to stop playing. Recently there had been symptoms of dyspnea and expectoration also. X-ray examination April 5 showed that the transverse measurements of the heart were increased. There was evidence of dilatation of the auricles. The outline of the heart was indistinct because of motion. The outlines of the various chambers could not be seen. The size and shape of the heart were consistent with rheumatic heart disease. She breathed rapidly at play and in her sleep.

She was normally delivered at full term and weighed five and a half pounds at birth. She was breast fed for no more than a week at the most. She had never done well on bottle feedings. She breathed and acted normally until the seventh day, when she was cyanotic for ten minutes. At five months she had measles. She was thought to have had tonsillitis several times. Her bowels had been irregular. At four years and three months she had chicken pox. At that time she was examined by the doctor in charge at her birth, who told her parents that she had heart trouble which might or might not give trouble later.

Clinical examination showed an underdeveloped and undernourished child sitting upright in bed crying, in no apparent physical distress. Skin pale, not cyanotic. Throat and tonsils injected, tonsils cryptic. Posterior cervical glands enlarged. Pulsation of the great vessels in the neck. The chest wall pulsated with each heart beat. Precordium bulging. Transverse diameter of the heart three and five-eighths inches. Left border five-eighths of an inch outside the apex. Supracardiac dullness two inches. Apex impulse of the heart seen and felt in the fifth space just inside the nipple line, two inches from midsternum. A palpable systolic thrill and a systolic murmur, loud and rough, heard best at the apex and along the bottom of the heart and the left sternal border. The murmur was widely transmitted over the precordium and the great vessels. Pulmonic second possibly reduplicated. No diastolic murmur. The sounds were of fair quality, although the aortic second sound was rather distant and indistinct. Blood pressure 94/70. An electrocardiogram showed sino-auricular tachycardia, rate 150, slight left axis deviation. Abdomen depressed. Extremities and reflexes normal. No clubbing of the fingers.

Amount and specific gravity of urine not recorded. Other findings normal. Blood: 17,200 to 13,900 leukocytes, polymorphonuclears 60 per cent, hemoglobin 65 to 105 per cent (corrected Sahli), reds 3,344,000. Wassermann and Hinton tests negative. Tuberculin 1 to 1,000: human positive, highly inflamed; bovine positive. One throat culture doubtful for Klebs-Löffler bacillus, two negative.

Temperature 96.7° to 101.4°. Pulse 101 to 148, with one drop to 60 the morning after admission. Respirations 28 to 68.

The heart action was 140 at admission and continued rapid, 120. The murmur was of maximum intensity at the apex. There was comparatively little enlargement of the heart even though there was bulging of the precordium.

The night after admission the child had numerous coarse râles throughout both chests, coughed considerably and had a temperature of 101°. By April 25 the general condition seemed improved. The upper respiratory infection seemed to be entirely gone. During the next two days the temperature was again elevated with nothing but upper respiratory infection to account for it. For the next three days the temperature and respirations were normal. The blood pressure was 100/75. The murmur continued unchanged. May 5 she had acute follicular tonsillitis. By May 12, although this had cleared up, she continued to run a rapid pulse and an irregular temperature. May 19 she was discharged improved.

History of interval. After leaving the hospital she had very poor care. She was not kept in bed according to instructions, but was allowed to be up and about part of the time. May 31, twelve days after her discharge, the parents brought her to the Out-Patient Department, realizing that her condition was worse.

Second admission, May 31. On examination she was thin and wasted, sitting propped up in bed with respiratory difficulty, very weak, apathetic, sleepy and restless. Skin ashen pale. Face sunken. Slight cyanosis of the lips. Tongue showed a mottled, glazed white coat.

Mucous membranes high colored. Tonsils enlarged and cryptic. Some mucous exudate in throat. Teeth poor. Throbbing pulsation of the neck vessels. Venous hum and transmitted murmur audible in the neck. Chest expansion diminished. Respirations rapid, shallow and difficult, with occasional grunt. After ten or twelve rapid shallow respirations the breathing stopped for a few seconds. A few moist râles along the base of the left lung posteriorly. Heart enlarged downward to the fifth interspace and to the left half an inch outside the nipple line at the apex. No enlargement to the right. Slight enlargement at the base,—two inches in diameter. Rate generally 130, becoming more rapid when respiration increased. Heart beat almost disappeared at times. Visible apex impulse. Point of maximum intensity in the fifth interspace just inside the nipple line. Sounds loudest at the apex but more clear-cut at the third interspace just to the left of the sternum, varying in intensity. Slight thrill at the apex. Loud prolonged systolic murmur at the apex heard over the entire precordium and into the neck and left axilla, but loudest at the apex. No diastolic murmur made out. Radial pulse synchronous with apex beat. Blood pressure 86/56. Sounds at the base very faint. Aortic second greater than pulmonic second. Abdomen flat. Liver to the right palpable three finger-breadths below costal margin, somewhat tender. Tympany in pelvis. Slight edema of the ankles. No clubbing of the fingers. Knee jerks very weak but equal.

No laboratory data are recorded.

Temperature 100° to 98.7°. Pulse 130 to 122. Respirations 35 to 27.

The child was very fretful and restless. She was given two doses of digitalis grains iss. The cyanosis increased. The day after admission she died.

Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

I have been somewhat disappointed of late years in the amount and quality of the help that I have got from the X-ray in cardiac diagnoses. I think as compared with what radiology does for us in relation to the kidneys, the lungs and the stomach that what it does for us in the heart is relatively unimportant. We do not feel we are missing anything very important if we have no X-ray examination of the heart unless in one of those chests where we cannot tell by percussion whether the heart is enlarged or not and cannot feel the apex beat. Then if we want to be sure whether the heart is enlarged or not, X-ray is the best method. I think those cases are relatively few. X-ray does not help us to tell whether adherent pericardium is present or absent, or whether a valve lesion is aortic or mitral, or whether syphilitic aortitis exists. Those are problems which I have seen put to the X-ray man when the clinician was in doubt. I do not think I have seen the X-ray help in them. It does occasionally help in congenital heart disease. The unusual protuberance generally seen in the region of the left auricle, a protuberance greater than we see in mitral disease, does help us in congenital heart disease.

I do not know any way to be sure whether or not the attack of cyanosis mentioned in the past history was connected with the heart. A good many things can happen to a child, even a child with a normal heart, that can give short attacks of cyanosis.

As we go over these records here that phrase, "tonsils injected," seems to occur in almost every record of any man, woman or child coming into this hospital. It has practically no value. We see it in people who have sore throats; we see it in people who have not sore throats. I think it might just as well be left out.

I think it is fair to say that a palpable thrill is never present without organic heart disease. But there is almost no variety of heart disease that cannot have thrill. Hence beyond saying that there is organic heart disease, the thrill does not help us. The fact that it was widely felt tends to show that it was a forcible thrill. The most forcible thrills we feel come in congenital heart disease, just as the loudest murmurs we hear come in congenital heart disease.

None of that clinical examination helps us much that I can see toward diagnosis.

There is nothing in the urine or blood examination that helps me at all.

The tuberculin tests I think do not help us. It is pretty clear this child is suffering chiefly from heart disease and not from tuberculosis. The fact that tuberculous infection is present is only what we know about the vast majority of all children living in cities.

A temperature of 101° in a baby of this age means practically nothing.

With the type of patients who come to this hospital it is almost useless to tell a mother to keep a child of this age in bed. She cannot do it. You have to go yourself and see it done if you have any idea of having it done. It takes more time than the mother can afford to give presumably.

PHYSICAL EXAMINATION

I suppose that report on the face and throat means that she is dehydrated. There is not enough fluid put into her or she has been vomiting or has had diarrhea.

The fact that her breathing stopped for a few seconds is not of much significance in a child. Of course that makes us suspect Cheyne-Stokes' breathing. But some normal children have Cheyne-Stokes' breathing, especially if drowsy

or asleep. It has not the same significance in a young child as in an adult.

Presumably the apex thrill is slight now because the heart is less strong. The stronger the heart, other things being equal, the stronger the thrill. When the heart is weak, the thrill is going to be weak.

DIFFERENTIAL DIAGNOSIS

I do not think we need doubt that she died of heart disease. Whether there was any complication I will consider later; but the underlying trouble I think we ought to say is heart disease. Yet that is not so clear as it might be. There is no question about the murmur's being abnormal. But how much passive congestion she has is dubious. She has edema of the ankles, a few râles in the lungs, an enlarged and tender liver. But none of those things is of the degree you expect in a child who not only has heart disease but dies of heart disease. I have no doubt she has heart disease. I have some doubt as to its being the chief or only cause of death.

What variety of heart disease could the child have? She certainly could have congenital heart disease. We have no negative evidence against there being murmurs present at the time of birth. The first time any examination was recorded a murmur was found, and the other facts are perfectly consistent with congenital heart disease. It usually gives a systolic murmur and thrill, with the murmur widely transmitted, as it is here. It usually is without distinct arrhythmia, as it is here.

What other type of heart disease might it be? Rheumatic. The chief point against that is the age of the child. It is not common to see rheumatic heart disease at this age, but it is not unknown. It has even been recorded in intrauterine life. So there is no age at which a child is too young to have rheumatic heart disease. It is true she has never had joint trouble. But that does not in any way exclude rheumatic heart disease. It may be primary in the heart and only considerably later show at all in the joints. She certainly had acute tonsillitis. She had one attack while she was here.

If it is rheumatic heart disease it is presumably mitral. In young children mitral disease which gives only a systolic murmur is not at all uncommon. But if she had mitral disease one would suspect right rather than left axis deviation by electrocardiogram and one would expect more passive congestion of the lungs.

Is there any other type of heart disease we ought to consider? Syphilitic heart disease does not occur in a child of this age to any extent at all. It certainly is not hypertensive. Should we consider bacterial endocarditis? We have little evidence of it. First, this is a rare disease at this age. We have some fever, it is true, but not much. We have no distinct leukocyte change. The leukocytes are practically normal for a child of this age. We have no evidence of embolism and no positive blood culture. I should say that bacterial endocarditis is improbable but not impossible. It is more likely to be congenital heart disease than any other type, with rheumatic as a second.

What else may have contributed to her death? She certainly is badly nourished. Any badly nourished child is likely to have bronchopneumonia. If you go over the autopsy records of children you get bored with the monotony of that finding. Any child who dies of anything has also a bronchopneumonia. It is a pretty safe bet when a child dies to say that he has bronchopneumonia. The question is, is there clinical evidence enough? The physical signs are not distinctive. They are such as might go with bronchopneumonia or without it. There certainly is no evidence of a large area of pneumonia or empyema or pericarditis, the complications one is looking for in children's heart disease.

Have we anything more to say about tuberculosis? She had a positive tuberculin reaction. She presumably has some tuberculosis. Did that have anything to do with her death? I do not see how we can say that it did or deny that it may have. But I do not believe tuberculosis was an active factor. The only type that could contribute much to her death would be miliary, and that usually gives meningeal symptoms as part of the picture. On the whole I think we should say that she has a tuberculous infection, but not the disease tuberculosis in any active form.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Congenital heart disease.
Probable rheumatic mitral disease.
Congestive heart failure.

DR. RICHARD C. CABOT'S DIAGNOSIS

Heart disease, probably congenital, possibly rheumatic.

ANATOMIC DIAGNOSES

1. *Primary disease.*

Congenital anomaly of the heart: interventricular septum defect.
Acute endocarditis, probably rheumatic.

2. *Secondary or terminal lesions.*

Bronchopneumonia.

3. *Historical landmarks.*

Tuberculosis of mesenteric lymph nodes.

DR. TRACY B. MALLORY: This child had a congenital heart anomaly consisting of an interventricular septum defect about one centimeter in diameter. The right ventricle was almost as large as the left. It is rather hard to imagine why they did not get right axis deviation. The

degree of right ventricular hypertrophy was much greater than I have ever seen in mitral disease. There was a slight degree of endocarditis, minute granules typical of what we call acute rheumatic endocarditis, on the mitral and tricuspid valves. They were too recent to cause any valvular dysfunction. Of course where an acute rheumatic endocarditis is present a myocarditis becomes very probable.

There was a slight degree of bronchopneumonia and the mesenteric lymph nodes were uniformly somewhat large, three of them definitely caseous, the tuberculous focus responsible for the positive tuberculin tests.

There was only a slight degree of passive congestion.

DR. CABOT: So that in your opinion the heart was not probably the main cause of death?

DR. MALLORY: I think it is possible that acute endocarditis on top of the old lesion made a rather rapid failure at the end.

DR. CABOT: When we come out partly wrong, as we do in this case, we then have to ask ourselves whether we made any mistake that we can avoid again. We considered acute endocarditis, but I do not believe vegetations of this size are capable of sure diagnosis by any method we yet have. I do not see any particular lesson we can gain from this case. General principles are going to guide us wrong again as much as they did here.

I neglected to state what type of congenital heart disease it was. But I should not have got anywhere if I had tried to. It is useless to say more than "congenital heart disease". The different varieties cannot be distinguished clinically where cyanosis and clubbed fingers are absent.

A STUDENT: Was there any myocarditis?

DR. MALLORY: I have not seen the sections, but I should expect to find it.

A STUDENT: Should you not expect diastolic murmurs with interventricular septum defect?

DR. CABOT: No. Usually we get only a systolic. There is not pressure enough to produce a diastolic murmur. We may get just the same sort of murmur with any one or any combination of other congenital lesions.

A STUDENT: What part did the throat condition play in the downward course?

DR. CABOT: It probably played a considerable part. We naturally connect that with the acute endocarditis which Dr. Mallory feels must have been a large factor.

A STUDENT: How extensive was the bronchopneumonia?

DR. MALLORY: There were just a few small areas in the borders of each lung.

A STUDENT: What organism did cultures show?

DR. MALLORY: We got none.

DR. CABOT: That is why you call it rheumatic?

DR. MALLORY: Yes.

A STUDENT: What is the cause of the anemia?

DR. CABOT: It is doubtful whether we can call it anemia. I think they ought to have found a little more than this to call it anemia. I should say the child never got fairly started on her adult blood picture. How much she is below that I cannot say. I do not think it is much.

CASE 15452

UNEXPLAINED HEMATEMESIS

SURGICAL DEPARTMENT

First admission. An American steamfitter fifty-eight years old was sent in by a physician July 3, nearly three years before his final admission, for study of anemia. The patient's own chief complaints were epigastric pain, rapid heart and weakness. He was so deaf that the reliability of the history is doubtful.

A year before admission he began to have a dull ache in the left epigastrium extending to midepigastrium and radiating to both lower chests, more often to the left. It occurred two or three times a week, usually about half an hour after eating, and was relieved by hot water and soda. It was accompanied by eructations of gas with partial relief. When untreated it usually lasted two hours. By regulating his diet, especially by avoiding greasy food, he found the attacks were infrequent and not troublesome. Seven months before admission he found on getting up in the morning that his heart was very rapid. He had no other cardiac symptoms. He had, however, often been dizzy in the past seven months. He stopped working for three months. The attacks of palpitation continued to occur at irregular intervals with and without exertion and excitement. He was very weak during the attacks and sometimes in the intervals. In February he came to the Out-Patient Department. Examination showed the heart not enlarged, action slow, an apical systolic murmur. An electrocardiogram showed normal rhythm, rate 70. X-ray showed no evidence of organic disease of the stomach or duodenum. Basal metabolic rate -8. Blood: reds 5,070,000, slight variation in size and shape. In April he returned to work for six weeks. Then he had to stop on account of weakness and palpitation. For several months the attacks of epigastric pain had occurred after nearly every meal. During the past six weeks he had had frontal morning headaches, relieved immediately on lying down. Two weeks before admission these stopped. He had recently noticed black stools and he thought some red ones. He had had no colic, vomiting, hematemesis, nausea or jaundice. He had some gas. He had lost possibly ten pounds since the onset. For the past three weeks he had felt better.

His family history is unimportant. His past history shows gonorrhea thirty-five years before

admission, with no recurrence, many boils lanced eight years before admission, and "catarrhal deafness" for six years.

Clinical examination showed an obese man lying quietly in bed in no apparent distress. Skin and mucous membranes somewhat pale, with only a suggestion of lemon yellow tinge. Heart moderately hypertrophied. A soft systolic murmur at the apex, transmitted to the axilla. Blood pressure 160/90. Abdomen soft; no spasm, tenderness or masses. Left indirect inguinal hernia and absence (congenital?) of left testicle. Rectal examination negative.

Urine: amount not recorded, no abnormalities except 2 to 5 leukocytes at 1 of 4 examinations. Blood: 4,900 to 7,100 leukocytes, 76 to 60 per cent polymorphonuclears, hemoglobin 50 to 70 per cent, reds 1,850,000 to 3,900,000, one smear normal, one showed slight achromia, a third some poikilocytosis with an occasional tailed cell, marked anisocytosis with many macrocytes, platelets reduced little if at all. Wassermann negative. Fasting contents of stomach: 10 c.c. cloudy grayish sour material, free HCl 4, total HCl 32, guaiac negative. Test meal: 22 c.c. similar material, free HCl 32, total HCl 52, guaiac negative. Stools negative.

X-ray examination with a barium enema showed no evidence of organic disease of the colon. Examination of the chest showed the lungs clear except for several calcified glands at each hilus. The cardiac shadow was slightly increased both to right and left. (Right 5.7 cm., left 9, chest 26.)

Pulse 100 at admission; otherwise chart not remarkable.

The patient did extremely well on the Minot-Murphy diet. He had no attacks of palpitation or other symptoms. July 14 he was discharged.

History of interval. After leaving the hospital he continued to do very well on the liver diet, though he still had attacks of palpitation. He was troubled with gas by mouth and by rectum, often accompanied by dull burning substernal pain usually occurring at bed time, never severe, relieved by belching and soda. Occasionally he induced vomiting for relief of gas. For three months before his second admission he had felt in relatively excellent health except for inconvenience from his inguinal hernia. Three weeks before his second admission this became irreducible and very painful.

Second admission. December 6, seventeen months after his discharge, he reëntered.

He was now well nourished, not obese. The hernia was successfully reduced on December 7. He made a good convalescence until December 24. Then he vomited coffee grounds material and passed tarry stools for the next four days. On examination December 28 his skin and mucous membranes were pale. Rectal examination showed tarry stools which gave a strongly positive guaiac.

Laboratory data after December 28. Urine: amount not recorded, no abnormalities except rare leukocytes at four of five examinations. Blood December 28: hemoglobin 55 per cent, reds 2,208,000, no achromia (reds showed more hemoglobin than normal), marked anisocytosis with scattered macrophages, moderate poikilocytosis; intense polychromatophilia, no stippling; 10,000 leukocytes, 70 per cent polymorphonuclears, numerous young polymorphonuclears and others with multilobular nuclei; reticulated cells 13.1; platelets appeared normal. Clotting time 4½ to 5 minutes. Fragility test began .46, complete .38. Non-protein nitrogen 31 mgm. January 9 hemoglobin 65 per cent, reds 3,800,000, slight achromia and variation in size, a few diffuse basophilic cells, 6,500 leukocytes, a few immature polymorphonuclears, platelets normal, reticulated cells 1 to 2 per cent.

Chart after December 28 not remarkable.

December 31 the stools gave a very strongly positive guaiac. The patient was symptom free. however, and after January 1 the stools were negative. January 12 X-ray showed a large accessory pocket arising from the posterior gastric wall just off the lesser curvature. The rest of the stomach and duodenal wall were normal. At twenty-four hours the head of the barium column had reached the rectum; the pocket in the stomach wall remained filled.

The patient continued to gain. A surgical consultant advised postponing operation as long as the patient was comfortable on medical treatment. January 23 the patient was discharged.

History of interval. He kept faithfully to the régime prescribed, gained in strength and weight and was symptom free for the first seven months except for gas and sour eructations. Seven months before his third admission he began to have cramp-like pains in the midepigastrium, constipation and gas. X-ray showed a "pocket in his stomach." Four weeks before readmission he began to have slight cramp-like, gnawing pain in the epigastrium coming several times a day without relation to meals and lasting only a short time. April 21, after feeling very well, he had itching over the whole body followed by a chilly feeling, then by hot flushes. Later in the day he passed dark red stools.

Third admission. April 23, fifteen months after his discharge, he reëntered.

On examination he was still well nourished, though he had perhaps lost some weight. The arterial walls were palpable and tortuous Blood pressure 155/98 to 127/78. The fundi showed arteriosclerosis.

Before operation urine much as before, hemoglobin 50 to 60 per cent, reds 2,840,000 to 3,850,000, moderate achromia, platelets normal, 8,800 leukocytes, 72 per cent polymorphonuclears. Hinton negative. Stools tarry at three of seven examinations, guaiac strongly positive at four, positive at two more.

Before operation chart normal except for a

rise in temperature (100°) and pulse (111) on April 29.

The patient had no symptoms. The stools continued however to show a positive guaiac. May 11 operation was done. The wound showed some serous discharge and was somewhat slow in healing. Following a sneeze it was found wide open. During anesthetization for resuture the patient strained, forcing out loops of small intestine. He developed a pneumonia. The wound showed pus. June 7 he died.

Discussion

BY CHESTER M. JONES, M.D.

NOTES ON THE HISTORY

Certainly from the patient's chief complaints the rapid heart and weakness might perfectly well be explained by anemia. The deafness we had better leave out of the picture for the moment, though it may be of importance as we go farther.

His symptoms are not particularly characteristic of any gastro-intestinal disease, I should say. The fact that the pain has definite relation to intake of food suggests peptic ulcer, but it is a little odd to have peptic ulcers cause pain today and not tomorrow. It is rather an atypical story for peptic ulcer. Frequently ulcer patients do have pain only at one hour of the day, sometimes in the afternoon, more often after the evening meal.

The presence of eructations of gas is of no importance. It is simply a habit or a reflex symptom which is associated with intragastric or other gastro-intestinal diseases, and is not characteristic of any one of them. Relief by soda is not diagnostic. It is true that most patients with peptic ulcer are relieved by taking soda. It is true that most stomach-aches are relieved by soda whether there is too much acid or too little acid. It means that the symptoms are caused by something going on in the stomach; but that in turn may be an intragastric or a reflex disturbance, and the effect of soda is not primarily so much one of changing the acidity of the stomach as of altering its tone or relaxing spasm. The real action of soda is not understood precisely.

On leaving out greasy food he was better. We know that with too high a fat intake we slow up the progress of food through the stomach. He had better gastric motility in the absence of too much fat. I do not think there is anything diagnostic in that.

Vertigo and tachycardia can certainly be explained entirely on the basis of anemia. We have a right to assume he is anemic, for his doctor sent him in because of that. Any cardiac symptoms can be explained on that basis.

His stopping work presumably means that the trouble was fairly severe.

I am inclined to doubt the statement that the attacks of palpitation had no relation to exertion or excitement. Probably if we had questioned very carefully we should have found that both exertion and excitement did cause palpitation and probably nothing else did produce these attacks, unless he had true paroxysmal tachycardia. We do not know many details about these attacks, and it is hard to do more than speculate.

The heart examination in the Out-Patient Department shows no real evidence of heart disease at all. On that and the electrocardiographic report I should say we can rule out the heart as the essential factor in this diagnosis.

The X-ray report may mean one of two things. It may mean no disease and it may mean the disease was not found. I think we can say there is no obvious gastric pathology, because apparently this is an entirely negative X-ray report. There is no suggestion of some of the significant findings frequently made on fluoroscopic examination. There is nothing to suggest disturbance of the gastric function.

The basal metabolic rate is within normal limits.

The blood count shows no anemia unless the hemoglobin was down, and we have no data as to that. Certainly nothing but secondary anemia could be present if that red count is correct.

After three months' rest he returned to work, but again the same symptoms caused him to leave work.

I am beginning to question the correctness of that red count, because he certainly had symptoms of anemia and we have his doctor's statement that he has anemia. In the rush of examinations in the Out-Patient Department it is not always possible to do accurate work.

His weakness, palpitation, headaches and so on may be associated with anemia. We do not need to assume any intracranial lesion. Possibly he rested a little more and so stopped the headaches.

A true story of tarry stools is very significant. The first thing to find out is if he had taken iron or bismuth before. He was treated in the Out-Patient Department, but probably was not given iron because they found a normal red count. His own doctor may have given him iron. But I take it he is just now being treated in the Out-Patient Department and the black stools are probably not due to iron. In the absence of those two drugs and if the stools are really black—most stories of melena are incorrect—then we have to assume he has been bleeding from some part of the gastro-intestinal tract reasonably high up. One can have red blood, fairly dark red blood, from lesions well up in the gastro-intestinal tract, even bleeding ulcer. But when we have really red blood in stools it is fair to say the lesion is usually low down in the gastro-intestinal tract. If it is in the rectum then the blood is bright red and is probably around the stool, not mixed in it. If the bleeding is from the colon we may well have red stools. For instance carcinoma of the colon tends to give red blood in the stools. Small in-

testinal lesions may also give fresh blood or tarry stools. In this case the stools make us think the lesion is fairly high up, presumably in the small intestine or in the vicinity of the stomach. The thing to remember is not that we get red blood in the stools from lesions low down, but that rarely it can occur when the lesion is in the stomach, but usually with loose and frequent movements. The fact that he had no hematemesis does not prove that the bleeding did not come from the stomach.

Gas is of no particular importance so far as hematemesis is concerned.

That loss in weight may be entirely due to the fact that he has been afraid to eat, or it may mean that in spite of a good food intake he has some process causing loss of weight. Usually in these cases the patients limit their diet voluntarily because they think certain articles of food tend to produce symptoms. At the age of fifty-eight we have to pay great respect to loss of weight.

My feeling is that part of the apparent improvement is due to the fact that he rested before he came into the hospital.

All we have up to the second admission is the story suggestive of gastro-intestinal hemorrhage with associated symptoms of anemia. I think the gastro-intestinal symptoms somewhat suggestive of ulcer. They have gone on for a year. Most of our patients with cancer of the stomach present a story of only a few months. This is rather longer than the history of the average case of cancer of the stomach. It is a little unusual in cases of peptic ulcer to have symptoms begin at the age of fifty-seven. We still have to consider two causes of tarry stools, polyposis, a very rare disease, and diverticulitis. Diverticulitis is very rarely associated with hemorrhage. Usually there is hematemesis with cirrhosis before we get tarry stools. But that has to be considered as a cause of the anemia which I believe he has.

The "catarrhal deafness" for six years presumably is due to a catarrhal condition of the Eustachian tube, one of the most common causes of deafness. Evidently he had been told it was that and he seemed to feel it a reasonable explanation. I do not believe we have to consider less common causes, such as syphilis or any nerve disease.

The very fact that they mention lemon yellow tinge indicates that the person who made the physical examination was thinking of pernicious anemia. Such a remark should not be disregarded.

We do not know what the subsequent blood pressure readings were. There is not hypertension apparently, although the blood pressure is rather high for a patient with marked anemia. He may well have at the age of fifty-eight a certain amount of hypertension and evidence of arteriosclerotic heart disease. At the same time I believe cardiac disease cannot be of more than passing interest. I do not believe it is important in considering the diagnosis.

The abdominal examination does not help us at all one way or another. Frequently we do not feel cancer of the gastro-intestinal tract, and still more frequently we do not feel masses when a patient has an indurated ulcer. It is very rare to feel a mass in relation to peptic ulcer. Occasionally it can be felt. This man is obese, and abdominal examination cannot be entirely relied upon.

The left testicle may be absent or undescended. He has had no operation for removal of the testicle.

The physical examination does not help us very much. He is a little pale and there is a slight suggestion of lemon yellow tinge. The urine is essentially negative. He has an anemia; there is no question about that. His red count cannot have increased that much in eleven days unless he really had pernicious anemia and responded most promptly to the liver diet. Perhaps the blood counts are not entirely correct because of variations in blood-counting pipettes. It is fair to say this man has an anemia. I do not know how much it is, but it is fairly severe, somewhere near 3,000,000 with a hemoglobin of about 60 per cent. That is certainly not a very low color index, neither is it a high one. There is no leukocytosis, and the differential count is normal. A diagnosis of achromia based on the smear is a very dangerous diagnosis to make, because in making a smear we sometimes squeeze the red cells too much, so that they look paler than they are. Those findings are entirely consistent with any chronic anemia, and we have every reason to doubt that Out-Patient Department red count. His doctor was probably correct, as doctors usually are when they send in patients with definite statements about them.

Macrocytes are supposed to be characteristic of pernicious anemia. If they were macrocytes I hesitate before saying the patient did not have pernicious anemia. We may have variation in the size of the red cells in any severe anemia. I am going to put a question mark after that blood smear. It is not sufficient to make a diagnosis on. We have been mistaken again and again when we thought pernicious anemia was the correct diagnosis, when autopsy showed cancer or syphilis. But this is certainly rather suggestive of primary anemia. The cells are very well filled with hemoglobin and there are cells described as macrocytes. The platelets are not reduced. Usually the platelets are definitely reduced in pernicious anemia. Another thing against it is the normal white count. Another thing is the almost normal polymorphonuclear percentage. In pernicious anemia, with a red count about 3,000,000 we should have a low white count with fewer marrow elements than are present here. It is fair to put a definite question mark against pernicious anemia.

The stomach analysis shows that there is no stasis here. Those are perfectly normal stomach contents. That is very definite evidence against pernicious anemia. We have only one case of pernicious anemia on record at this hospital with free acid in the stomach contents, and I believe such a diagnosis here is untenable.

He is not bleeding now. How many stool examinations were there?

MISS PAINTER: Two.

DR. JONES: He certainly is doing no more than oozing a little or he would have a positive guaiac in his stools.

The fact that the X-ray showed no evidence of organic disease does not mean that he has not got it.

The calcified glands are probably of no significance. I think anyone at fifty-eight might perfectly well have calcified glands.

The cardiac shadow is a little more than half the transverse diameter of the chest. It fits in with the percussion measurements, indicating slight arteriosclerosis and hypertensive heart disease. In my mind he has a secondary anemia associated with gastro-intestinal pathology. Secondary anemia does not give so rapid a response to liver treatment as primary anemia does. I do not know exactly what they mean by the fact that the patient did well on the Minot-Murphy diet. Probably they mean that symptomatically he did well. Possibly his anemia was somewhat improved. Did he have iron?

MISS PAINTER: There is no record of it.

DR. JONES: The best combination in secondary anemia is liver and iron, the latter in very large doses. He probably did not have it.

I think the diagnosis made at the first entry was probably questionable pernicious anemia. He left the hospital so far as I am concerned without a diagnosis except anemia, probably due to disease in the gastro-intestinal tract. I am sorry that they did not repeat the X-ray examination of the stomach when he was here at the time. They probably trusted to the findings in the Out-Patient Department and merely had a barium enema.

I think the attacks of palpitation after he left the hospital are partially due to the fact that the anemia is not entirely gone and probably because the cause of the anemia is not gone either.

Heartburn can be associated with no free acid in the stomach or with very high acid. It is not diagnostic of any particular disease. We do not know whether this came two hours after meals or began coming on immediately after he ate. The presence of heartburn makes us think of a gastric or extragastric lesion in the duodenum or possibly in the gall bladder. Disease in the gall bladder could not explain the bleeding.

We do not know what the character of the vomitus was. Probably it was regurgitation of small amounts.

He comes in five months after his discharge with most of his symptoms improved. The anemia probably is very much diminished. His gastro-intestinal symptoms are much improved. He comes in for another reason, namely, hernia. One wonders whether hernia alone caused the previous story. I doubt it.

The character of the vomitus on December 24 is definite proof that he has had hemorrhage from the upper gastro-intestinal tract. We assume this material came from the stomach and not from the lungs. Hemoptysis and the swallowing of blood may cause "coffee-grounds" vomitus. This story is quite straight. It was probably a real hemorrhage from the stomach or near by. It may be due to esophageal varices or to local gastric ulcer or to duodenal ulcer. As far as the blood is concerned any one of those lesions could cause the bleeding.

We now have all the evidence we need to be sure the previous story is correct.

All the blood findings can be explained this time on the basis of brisk hemorrhage. Brisk hemorrhage with a good marrow should give a rapid increase in reticulated cells. It should give polychromatophilia, should give normal or increased platelets, should give poikilocytosis. I do not think the blood findings mean anything more than a normal reaction to fairly brisk hemorrhage.

The clotting time is normal.

The figures for the fragility test are closer together than normal, as is frequently seen immediately after a hemorrhage.

I should say he has a good bone marrow, and his blood has come back as well as any patient's under such conditions might. There is no evidence here of pernicious anemia. I am still against such a diagnosis. Pernicious anemia does not give gastric hemorrhages.

Immediately in the vicinity of December 28 there was tachycardia possibly with some fever, which then dropped to normal with cessation of the bleeding.

That is the usual course of events; occult blood for a time after the hemorrhage ceases, then a return to normal stools.

Eleven days after he had evidence of no more bleeding he was X-rayed. That is a fairly safe time to X-ray after gastric hemorrhage. Possibly one might like to wait a little longer with a very sick patient. Dr. Hampton, will you demonstrate this X-ray film?

DR. A. O. HAMPTON: When the patient came in he was placed before a fluoroscope and a collection of barium was seen just below the diaphragm at the entrance of the esophagus into the stomach. It is of course unusual to see a collection of barium there. That roused interest. The patient was then given barium. It filled up a large pocket 2.5 centimeters in diameter connected by a small neck to the stomach.

DR. JONES: So there is a pocket there holding barium for a long time. I should like to ask

one question, Dr. Hampton. Should you say that with this pocket that remained full for twenty-four hours we could consider the diagnosis of diaphragmatic hernia? This man is at an age when diaphragmatic hernia may cause symptoms, sometimes with bleeding, although I think real hematemesis does not occur.

DR. HAMPTON: Yes; diaphragmatic hernia should be considered. It is too bad we did not have a repetition of his X-ray when he was in the hospital before. We might have seen the pocket the first time. Sometimes these pockets are so small that they fill with mucus and food and we do not see them until later, when they become larger. That may be what happened here.

DR. JONES: It looks as if he had a diverticulum of the fundus of the stomach near the cardia; or it could be a large gastric ulcer which has penetrated and walled off and has almost severed connection with the stomach. Is that the crater of a gastric carcinoma? Yes, possibly; but I think it is entirely unlikely. The presence of free acid in his stomach is against a diagnosis of carcinoma, though it does not disprove it at all.

The postponement of the operation indicates one of several things. The job was a very difficult one and the surgeon did not think the risk from postponement was sufficient to indicate immediate operation. That region of the stomach is a very difficult one to operate on. He thought that there was not any immediate danger, and that the patient could be tided along. He may have thought the patient had inoperable cancer.

The second admission was seventeen months after his first admission, in other words a year and a half. He then had a story of two and a half years of symptoms. The third admission was fifteen months after that. The interval between the second and third admissions was about fifteen months, a little over a year.

What régime was the patient following?

MISS PAINTER: He had a six meal bland diet.

DR. JONES: They thought he had an ulcerative lesion. Whether they thought it was peptic ulcer is another matter.

With any gastric lesion it is not uncommon to have constipation. That does no more than complicate the picture. It has nothing to do with the diagnosis.

The pain coming several times a day without relation to meals does not sound too much like ulcer. It sounds rather unlike it, I should say. So far I do not think we have definite evidence that this man had peptic ulcer.

I should say itching, chill and hot flushes were vasomotor reactions connected with hemorrhage. He had hemorrhage, and again we note the dark red stools.

Three years after he was first seen and four years after his story begins he comes in for his last admission. I should say that a four year story of this sort with remissions and relapses is very definite evidence against cancer.

NOTES ON THE PHYSICAL EXAMINATION

The blood pressure is about what it was before. It dropped a little later. That may have been due to hemorrhage. We have perfectly good evidence that he has sclerosis, and that fits in perfectly with the heart findings made before the operation. I do not believe it has any connection with the cause of his bleeding.

The smear is very much like the previous one. Apparently there is not so much evidence of bone marrow regeneration this time. The more chronic the hemorrhage, the poorer the marrow response becomes. The negative Hinton is evidence against syphilis and a syphilitic lesion as the basis of this picture.

We have gastro-intestinal bleeding as evidenced by a story of weakness, red stools, anemia, later tarry stools, and finally a strongly positive guaiac. Both of the latter findings are undoubtedly associated with hemorrhage and possibly some retention of old blood in the gastro-intestinal tract. Both will be associated with some rise in temperature.

DIFFERENTIAL DIAGNOSIS

It seems to me we have a good deal of evidence against pernicious anemia. One can get a picture of pernicious anemia with gastric bleeding with polyposis of the stomach. It is a rather rare condition. It is associated however with anacidity. There is a good deal of hydrochloric acid in the stomach. I think he has not got polyposis in the stomach or duodenum.

He can have logically one of two things—peptic ulcer which has been walled off and partially penetrated. Certain things fit in with this. The story is not atypical of ulcer more typical of a large diverticulum of the stomach which has become eroded. My diagnosis lies between those two diagnoses. He obviously has a large excavation connected with the stomach.

I do not think we need to consider other causes for hematemesis such as cirrhosis of the liver.

I should like to put in one more possible diagnosis, leiomyosarcoma of the stomach because of the ulcerative lesion. I should say leiomyosarcoma would fit in perfectly with the story. We have this excavation or diverticulum. So far as I know penetrating lesions do not occur in leiomyosarcoma. The duration of the disease and the clinical story would fit in perfectly.

I presume the operation was résection of the ulcerated cavity or diverticulum.

The slow healing of the wound is probably of no significance. We wonder when a wound heals slowly whether tuberculosis or syphilis is not present. This could be tuberculosis, but I am not going to mention more than the possibility. I do not believe it is syphilis. This man was obese, the kind of man who presents a rather

difficult surgical problem from the point of view of postoperative complications. He must have had a fairly large incision. We wonder why he coughed or sneezed. It may have been the start of a pneumonia. He may have developed pneumonia even before that. He has a peritonitis now without any question. I think we shall find he has hypostatic pneumonia, as so many elderly fat people with poor hearts have.

I think this patient will show simply pneumonia, peritonitis and a postoperative condition in the stomach, namely resection for a single ulcerative lesion, probably a diverticulum.

PRE-OPERATIVE DIAGNOSIS

Carcinoma of the stomach.

OPERATION

Paramedian incision to the left of the median line. The spinal anesthesia was unsatisfactory and it was necessary to supplement it with ether. On exploration the stomach showed no evidence of carcinoma or ulcer. The pylorus was unusually large, with very little musculature. At the cardiac end near the esophageal opening there was a small pouch. Both the greater and lesser omentum were extremely fatty, so that exploration was extremely difficult. There was no induration or tumor anywhere palpable. The liver was about half normal size. It was smooth in contour and normal in color. It showed no signs externally of disease. The left lobe was almost entirely missing, so that the dome of the diaphragm could be plainly seen with the central tendon. The spleen lay in close contact with the esophageal end of the stomach. The gall bladder was negative. Palpation of the entire length of the large intestine was negative except that there were hard fecal masses in the sigmoid. The small pouch at the cardiac end of the stomach did not seem to correspond accurately with the X-ray picture, as it was about the size of the last joint of the thumb and had a good sized opening into the stomach. There seemed no indication to open into the lumen of the stomach. The wound was closed without drainage

FURTHER DISCUSSION

He did have a diverticulum in the stomach. I think that is the reason for his bleeding. The variation from the X-ray pictures I think is very interesting. In the first place the difference in size between the X-ray and what they found. In the second place he may have had some ulceration. The actual lumen connected with that diverticulum was very small at times and may have been very small when he was X-rayed.

DR. HAMPTON: According to the original note the diverticulum was 2.5 centimeters in diameter. At the second examination the pouch was much smaller. At the examination seven months later it could not be found at all. At the examination five months later it was a large pocket.

DR. JONES: In other words there was an inflammatory condition with a great deal of irritation around the opening of it. If there is a diverticulum in the stomach we very frequently find them elsewhere. I cannot explain the size of the liver.

There is only one other possibility, that is the man may have a diverticulum which completely misled the surgeons. That is, he may have had cirrhosis of the liver with esophageal varices to account for the hemorrhage, and the diverticulum may have been put in to make it hard. It is usually true that diverticulum does not cause symptoms. The finding of a diverticulum is usually an X-ray finding. But I am not sure that in individual cases it can not cause symptoms. A pocket like this might well cause the symptoms of which he complained. I am under the impression that gastric diverticula bleed more frequently than those found in the large bowel. In this case we may have to make two diagnoses. I should say he certainly has diverticulitis of the stomach. He may have cirrhosis of the liver with esophageal varices.

DR. CHESTER M. JONES' DIAGNOSIS

Diverticulitis of the stomach.

Possibly cirrhosis of the liver with esophageal varices.

ANATOMIC DIAGNOSES

1. *Primary disease.*

Duodenal ulcer.

2. *Secondary or terminal lesions.*

Diverticulum of the stomach.

Broncho-pneumonia.

Hypertrophy of the heart.

Arteriosclerosis.

DR. TRACY B. MALLORY: There were two important findings, one of which was correctly diagnosed as a diverticulum of the stomach. This, I believe, however, was not the cause of the symptoms, but a large penetrating ulcer on the posterior surface of the duodenum, diving into the head of the pancreas. It was entirely on the posterior surface. Even at autopsy, palpating the duodenum from the outside I was unable to feel the ulcer. I did not find it until I actually opened up the duodenum.

So far as the surgeon's note on the size of the liver is concerned, certainly thirty per cent of surgeons' notes on the condition of the liver at operation are entirely wrong according to post mortem examination. I very seldom trust them. It was a perfectly normal liver weighing 1700 grams. I do not think the surgeon was to be blamed in not feeling that ulcer in the duodenum, because it was on the posterior surface, practically buried in the head of the pancreas. There were a great many old adhesions, one of

which ran from the transverse colon down into the inguinal ring. Another ran from the apex of the diverticulum to the lower pole of the spleen. So that it is probable that this is a so-called traction diverticulum.

His heart was hypertrophied, 420 grams, slightly above normal. He had a moderate degree of arteriosclerosis and some scars in his aorta that were suggestive of syphilis. He had a hypostatic pneumonia and a local peritonitis just beneath the operative wound.

The kidneys were practically normal. They showed a relatively slight degree of arteriosclerosis.

There is one statement of Dr. Jones' that I disagree with, in reference to leiomyosarcoma In a number of cases on record, which I have been looking up recently, diverticula were simulated by deep ulceration of an external myomatous tumor of the stomach.

DR. JONES: A surprising thing in this case is I think the fact that this man had such unusual pain, and that it was not referred to his back. When there is a duodenal ulcer, perforated, adherent in the region of the back, there almost always is pain in the back. This pain was epigastric, if anything higher up. It is strange he did not have more striking pain of penetrating ulcer. One thing we are sure of in the hospital is that a penetrating posterior duodenal ulcer is a difficult one to diagnose. The diagnosis usually is made because of the patient's symptoms in the face of the negative X-rays. We have had several cases lately in which the X-ray was negative or only suggestive of irritation in the duodenum, yet the patient's pain was out of proportion to the findings. There may be no tenderness on examination even with ulcer that is penetrating, or going on to perforation. It is surprising how little one can find in this type of ulcer.

There were no other diverticula, were there?

DR. MALLORY: No.

DR. JONES: How common is it for a diverticulum of the stomach to bleed? The ones in the colon bleed only on rare occasions.

DR. MALLORY: I do not know anything about it. This is the first case I have seen.

DR. JONES: We usually say diverticula do not bleed.

DR. YUSABURO NOGUCHI ASSERTS HE CAN REMOLD MAN

According to a statement in the daily paper Dr. Yusaburo Noguchi, Japanese biologist and experimental zoologist and head of the Noguchi hospital in Beppu, Japan, has claimed that after fifteen years of experimentation he had developed a technique for changing racial characteristics of human beings, even to the pigment colorings. He has recently visited this country after a period of study of the jungle peoples of Brazil working under a grant from the Japanese government.

Dr. Noguchi, although no relation to the late Dr. Hideyo Noguchi who died while working in Africa on yellow fever, was a classmate of his famous co-patriot many years ago.

By electrical nutrition and glandular control, Dr. Noguchi said he and his associates believed they could at will change an Indian to a darker color, and with the physical characteristics of a negro, or mold a Japanese so that he would have the same appearance as the Caucasian. The changes have been completed after the proper stimulation of certain gland secretions and the artificial diminution of others, beginning when the patient was a child.

Through an interpreter the biologist made it clear he could soon virtually transform an infant to order, making it tall or short, broad or slender, dark or light.

Not only in the physical side, but in the mental as well, Dr. Noguchi's experiments have proved successful. Underdeveloped children, some with distinct mental deficiencies in childhood have been changed, to grow to normal youth, he declared.

TULAREMIA FOUND IN MANY SPECIES

Reports of studies by Dr. R. G. Green and E. M. Wade of the University of Minnesota and the Department of Health tend to show that in addition to infecting rabbits, tularemia may also affect cats, muskrats, pigeons, pheasants, grouse and quail.

This increased number of possible hosts of this disease adds to the possibility of danger to human beings.

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THE NEW ENGLAND MEDICAL CENTER

A VISION of the future and an ideal of service is inspiring the consolidation of the Tufts College School of Medicine, the Boston Dispensary and the Boston Floating Hospital into a great New England Medical Center to be erected in the heart of Boston. Each is now a unique institution with a splendid record of service behind it; united, and with the same ideals of service to carry them on, they will become a power in medical progress.

The notable fact about this amalgamation is that no orthodox hospital and teaching center is being contemplated. It will not tread the well worn path of modern medical progress, but elects to forge ahead in a new way and yet in an old way, and certainly in an exceedingly useful way. Science and investigation have been well served in the medical advance of latter years, but many have sensed the danger that has resulted from this—the elevation of research and the laboratory to the position of leadership, and the relative neglect of the direct service to mankind which should be the ultimate goal of all advance.

We have heard much about the decline of the general practitioner and the abandonment of the rural districts to their fate, but very little has been done about it. The New England Medical Center, so far as it lies within its power, is pledging itself to rectify this evil, for its announced double purpose is to train family doctors for the smaller communities of New England and to provide more adequate relief for needy sick men, women and children. To serve the first of these purposes it will, in addition to training two hundred and fifty third-year and fourth-year medical students yearly, give informal graduate instruction to older practicing physicians, enabling them to come to the center from time to time for study and research.

The new Center will be erected at the present location of the Boston Dispensary on Bennett Street. Here the Boston Floating Hospital will build a forty-bed land hospital, the Boston Dispensary will remodel and enlarge its quarters, and a third building will be constructed jointly to provide a center of clinical training for the Tufts College Medical School, additional clinics for the Dispensary, and a dormitory for nurses.

The three institutions will continue to maintain their separate identities under the new arrangement and under a joint administrative board, but they have wisely appreciated the fact that in union there is strength.

CACHEXIA IN CANCER

THE most time honored symptom of cancer is the striking wasting that may appear to so marked a degree. This cachexia is frequently quite out of proportion to the amount of tumor tissue present, and at times the rapidity of its development is striking in the extreme. Again, some cases, even with widely disseminated malignant disease, die without any evidence of wasting.

The not infrequent occurrence of fever[1] in malignant disease is probably due to the autolytic products from the necrotic portions of the tumor tissue. While the kidneys in many cases of malignant disease show evidence of a tubular nephritis, it has not yet been definitely proved that this injury is due to direct effects of products of the tumor itself. It is, of course, inevitable that metabolic products formed by tumor cells should find their way into the circulation and so have an opportunity to act on the normal body cells.

Bierich[2] believes that the lactic acid produced in relatively large amounts by malignant tumors may be one of the substances favoring the development of cachexia. The relatively greater frequency with which cachexia appears in ulcerated tumors with secondary infection than in uninfected tumors even when the mass of tissue may be relatively large suggests that the prod-

ucts of bacterial metabolism acting either on tumor tissue itself or on the abnormal metabolites of tumor tissue may be important factors.

Clinical research on cancer patients might well be directed toward this common but yet obscure phenomenon.

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THE DOCTOR'S SADDLE BAG

THE SPECIALIST

INTRIGUING as the idea may be to those who are appreciative of native humor, and at the same time acquainted with contemporary literature—and the list must be a long one—our present subject bears not even a remote relationship to a current little book of well deserved popularity. We are, it has been remarked often and tritely, living in an age of specialism, and if the Cape Cod carpenter, in his simple way, wishes to follow the trend of the times, enough precedents have been established for him. The plumber cannot bore a hole in a board to run his pipe through; the furnace expert cannot fix a leaky faucet; the stone mason cannot lay a brick without placing himself in jeopardy of penalty, and, to bring the subject close to home, I but recently heard of an able psychologist who was warned by the psychiatric union that he must cease using a stethoscope if he wished to continue in the ranks of that select body.

To the way of thinking of some of us, specialism may be carried too far. When I find a painter who can pull a tack and putty the hole before he paints over it, I give him my confidence even if he has no union card in his pocket. When he patches the plaster before he hangs his paper I recognize him as a true craftsman even if the plasterers' guild would set their dogs on him, and when I meet face to face a carpenter who can point a chimney and set a pane of glass, and is not averse to doing so, I fall on his neck and offer him the best spare bedroom as a permanent home.

I like the country use of the word "able." It signifies the ability to do things and to do them well; it means usually the ability to do a number of useful things well. It is a mark of respect and we admire those to whom it applies. In medicine the able general practitioner is the highest type which we have yet produced; next to him comes the expert in a given line. The term "specialist" I deplore. I may be unique in my reaction to it, but it grates on my sensi-

bilities. To me it implies one who, not being able, has found it advisable to restrict his activities to a field which he can hope to encompass. There are those, also, who use its cloak more selfishly; it enables them to work less, to charge more, to exploit the false idea of ability which the public has read into the term.

No; specialization may be the coming thing in business and the trades, but it has been long enough in medicine to have gathered to it a rather unpleasant connotation. We have all more or less familiarity with a breed of patient which requires that its ills be treated only by a specialist. Their mental processes do not carry them to the point of enquiring as to the *type* of specialist required. What they want is a general specialist with a fine office, a good car and a silk hat. They do not want a general practitioner, no matter how able he may be—nor does he want them.

There are various ways of becoming a specialist, a very popular one being first to pick the specialty, then to take an intensive course of training, allowing from six weeks to three months for the metamorphosis, after which the deed is accomplished. The most approved modern method, since life, alas, is short, is for the young man to continue in specialized hospital work for a varying length of time, until he begins to acquire a true proficiency in his chosen branch. He may thus, indeed, become more or less of an expert, but unless he is unusually sagacious he is very liable to be unfortunately one-sided. It is not necessarily his fault but it is certainly one of the drawbacks of specialism. The best of the specialists whom we have known have been recruited from the ranks of the able general practitioners. Beginning with a broad foundation, they have builded upon it, gradually, through special interest and increasing expertness devoting themselves more and more to that branch of work which they liked best and performed most ably. Sir James MacKenzie was of this type; a "specialist" only by force of circumstances; an expert because of hard work, ability, and a consuming interest.

A large part of the value of the expert—call him specialist, if you will, and you will be literally correct—lies in his function as a consultant. As the general practitioner has definite responsibilities to the public, so has the expert responsibilities to the profession. It has been rather cynically said that the chief duties of the consultant are to make a digital examination and console the family, and too often this is about all that can reasonably be expected of him, but on the whole his field of usefulness is a broader one. It is his privilege and his responsibility to hold up the hand of the family practitioner, to bring to his assistance the most recent knowledge and the most advanced technique. The fruits of his studies and the results of his labors are ours to make use of. If he is faithful to his trust he cannot be one who has raised himself to a position of ease and opulence, for by his very excellence he has made himself the servant of those who have need of him. His rewards will be great—he cannot help that—but not the least of them will be the consciousness of the service which he is rendering.

We have recently lost by death one who was the ideal expert-consultant, and I believe he disliked the term "specialist" as much as I do. He was a humanitarian, and he felt keenly the responsibilities which he owed to his colleagues. He was always being called upon to give his opinions and his services, he was always available and he was always willing. He cared as little as any man I have ever known for the material rewards for his services. He was able and his ability was drawn upon.

MISCELLANY

DR. PHANEUF ATTENDS THE ASSOCIATION OF FRENCH SPEAKING GYNECOLOGISTS

Dr. Louis E. Phaneuf, Professor of Gynecology at Tufts College Medical School, attended the Sixth Congress of the Association of French Speaking Gynecologists and Obstetricians held in Brussels on October 3, 4 and 5.

Dr. Phaneuf performed two operations at the Medico-Chirurgical Institute—one, suspension of the uterus and appendectomy; the other, supravaginal hysterectomy for fibroids and appendectomy. He also read a paper on Complete Lacerations of the Perineum and Their Treatment.

Belgium, France, England, Italy, Switzerland, Greece, Spain, Roumania, Norway, Denmark, Holland, Brazil, Czecho-Slovakia and the United States were represented at the Congress.

TUBERCULOSIS ABSTRACTS

The importance of recognizing tuberculosis in its incipiency is repeatedly and deservedly emphasized. The alert physician, when he suspects the disease, makes careful search for lesions in the apex of the lung. The supposition is that the tuberculous process begins usually in the apex, from which focus it tends to spread downward to the lung generally. Several clinicians have recently called attention to a form of tubérculosis which begins in the infra-apical region and which spreads rapidly from there to other parts of the lung, including the apex. Max Pinner of the William H. Maybury Sanatorium, Detroit, has studied this type of lesion in 200 cases and presents his conclusions in this number of *Tuberculosis Abstracts*, a more complete discussion of which appeared in the *American Review of Tuberculosis*, February, 1929.

ACUTE SUBAPICAL TUBERCULOSIS

Wessler, some years ago, pointed out in this country the frequency of the early appearance of a subapical infiltrative process, the gravity of which, as regards progressive disease, was quite clearly shown. More recently, certain German workers, notably Assman, Redeker, Romberg and Ulrici, have pointed out that much of the tuberculosis seen in adults is the result of bronchogenic reinfection, either endogenous or exogenous, with a small tuber-

culous bronchopneumonic focus located anywhere in the lung, but most frequently in the infraclavicular region. This type of the disease comes on suddenly with symptoms that are grippe-like or catarrhal. Variations of this conception have been presented by different observers, but most of them agree that it is this infraclavicular infiltrative lesion and not the truly apical productive one, which is serious in terms of the development of progressive disease.

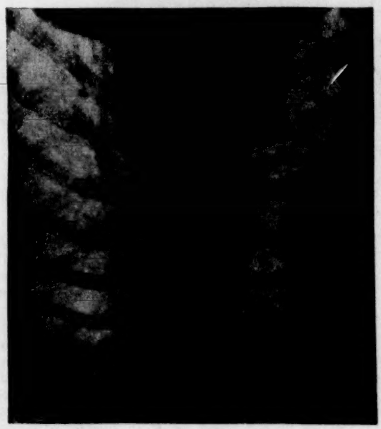

FIG. 1. This roentgenogram was obtained at a routine examination of employees. The patient at this time was in perfect health, and the roentgenogram does not show any evidence of tuberculous involvement of the lung, and in particular no apical foci.

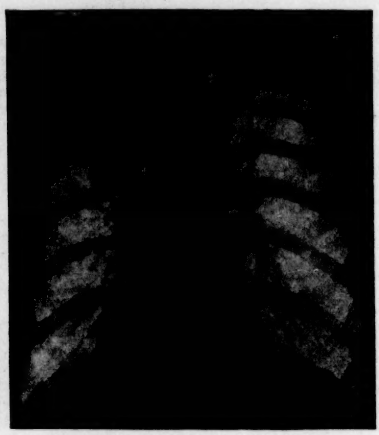

FIG. 2. The second roentgenogram of the same patient was taken seventy-eight days later and ten days following the onset of an acute grippe-like disease. It shows a rather extensive infiltrative lesion in the left infraclavicular region with beginning central excavation.

A study of the onset and type of lesion in two hundred cases has been made in an attempt to check the accuracy of the view that ascribes so much importance to the infraclavicular lesion.

Cavity formation was particularly observed as to the point of origin, migration, and especially the time of appearance in relation to onset. The rapidity of formation of cavities after onset, with the tendency to form in the subapical region and migrate upwards, was very striking. The x-ray was found to be the most useful in detecting the early development of the subapical lesion and bears out the value of using the x-ray as an adjunct to physical examination in every suspected chest condition.

TABULATION OF FACTS STUDIED

In these two hundred cases, a very careful history of the onset and course was taken, and every available x-ray of each case was studied. With this information tabulated, the following points stand out strikingly:

1. In pulmonary tuberculosis, sudden onset is not less frequent than is insidious onset.

2. Apical involvement is not characteristic of incipiency, but occurs in the majority of cases after the incipient stage has passed.

3. The extent of the lesion does not bear a direct relation to the duration of the disease. Of all patients reaching the far advanced stage, the majority do so within the first half year.

4. Cavitation is not a late occurrence; its frequency is nearly the same at any stage of the disease.

5. Apical involvement bears a close relation to the type of the clinical course.

6. An analysis of patients with pulmonary tuberculosis as to the two characteristics—"apices involved" and "type of onset"—leads to the differentiation of two different clinical types of disease. The one with apices free has a sudden onset with a bronchopneumonia (tuberculous) which usually progresses rapidly with acute exacerbations and the early development of advanced disease. The other type has an insidious onset, involves the apex primarily, and progresses very slowly, if at all.

Apical lesions as such are not always benign, as they often are the result of spread upward from a subapical lesion; however, this study would indicate that true apical tuberculosis seldom progresses caudalward to extensive disease.

SUMMARY OF CONCLUSIONS

1. Progressive and destructive pulmonary tuberculosis usually begins *suddenly*, with *exudative subapical* lesions.

2. Strictly apical productive tuberculosis is not, as a rule, the incipient stage of progressive and destructive pulmonary tuberculosis; it may precede the latter; but even in such cases, the latter starts usually as stated under 1. The rôle which apical tuberculosis plays in phthisiogenesis is rather insignificant as compared to that of acute subapical infiltrations.

3. Lesions far advanced as to extent, and excavations, frequently develop within less than six months.

4. Processes leading to active progression and to excavation are most frequently associated with acute symptoms.

5. Apical involvement, in the majority of patients, is not an occurrence of incipiency but a late development.

6. Diagnostic and therapeutic endeavors should be directed towards the acute subapical processes, and not only toward the insidious apical disease.

7. Physical signs and symptomatology, traditionally described as characteristic for "Incipient pulmonary tuberculosis," are misleading for the detection of truly incipient subapical acute processes.

Tuberculosis and health associations throughout the United States are now making plans for an early diagnosis campaign to be conducted during April, 1930. Billboards, posters, pamphlets, newspaper and magazine articles will emphasize the importance of discovering the early signs of tuberculosis in children.

THE GRADUATING EXERCISES OF THE TRAINING SCHOOL FOR ATTENDANT NURSES

The 11th graduation exercises of the Training School for Attendant Nurses were held Oct. 31, 1929 in the Copley Methodist Church at 180 Newbury Street, Boston.

The training school is carried on by the Household Nursing Ass'n at 222 Newbury Street, Boston. This class consisting of 96 women is the largest class ever graduated from the school. Rev. Guy H. Wayne, pastor of the church, opened the services with prayer. Mrs. C. Alexander Newhall, president of the Association, gave the address of welcome and introduced the speakers. Miss Elizabeth Ross, director of the Brookline Friendly Society, who was the first president of the Household Nursing Association was the principal speaker. Miss Ross, who spoke to the first graduation class of 8 women, now addressed the last and largest class.

Miss Ross said in substance that these young women were very fortunate in being so equipped that they might take their places in their communities in alleviating suffering in those families who could not afford the services of a graduate nurse. Although these girls are not registered nurses, they are sufficiently trained so that they can perform the common duties of a sick room, doing the necessary work for the sick person, cook for the family, maintain a pleasant atmosphere and attend to many other necessary tasks. Miss Ross stressed the point that a high ethical standard should be maintained by each graduate in order that she might be of the greatest service to her community.

After Miss Ross's speech the diplomas were distributed by Mrs. Eliot Wadsworth, chairman of the Attendant Nurse Committee. Miss Katharine Shepard is superintendent of the Household Nursing Association.

DEDICATION OF THE WILLIAM H. WELCH MEDICAL LIBRARY AND THE DEPARTMENT OF THE HISTORY OF MEDICINE, JOHNS HOPKINS UNIVERSITY

The dedication of the William H. Welch Library at Baltimore, Md., took place on the first day, October 17, 1929, of a two-day celebration at the Johns Hopkins Hospital and Medical School. On that day, in the beautiful hall of the new library building, there gathered around Dr. Welch many friends of long acquaintance, including Dr. Joseph S. Ames, President of the University; Dr. Harvey Cushing, who gave the leading address; Professor Karl Sudhoff of Leipzig; Mr. Abraham Flexner of the General Education Board; some of the trustees of the University, and the architect, Mr. Edward L. Tilton. Dr. Cushing spoke on the value of a central library to amalgamate not only the various parts of the Hospital and Medical School but also departments in the general University.

In the afternoon of the same day there was a conference in the School of Public Health on medical libraries and bibliography. Professor Sudhoff was one of the principal speakers. In addition, there were short talks by Colonel P. M. Ashburn, Librarian of the Army Medical Library, Washington, who spoke on "American Hospitals in the Revolution and the War of 1812"; Dr. Archibald Malloch, Librarian of the New York Academy of Medicine; Dr. William F. Francis, Librarian of the Osler Library, McGill University, Montreal; and Mr. Leonard Mackall from New York.

On October 18 there was the formal dedication of the chair of medical history, to be occupied by Dr. Welch. This took place also in the new library building. There were a number of speeches by Dr. Welch and others, including Professor Karl Sudhoff. In the afternoon there was a conference on medical history. Mr. Stephen D'Irsay, who will be associated with Dr. Welch in the department of the history of medicine, read a paper on "The Teaching of Medicine in the Early Universities". This was followed by brief communications by Dr. Edward B. Krumbhaar of the University of Pennsylvania, who spoke on Dr. John C. Otto of Philadelphia; Dr. E. C. Streeter of Boston, who talked on the new survey now being undertaken on the economic aspect of medicine, part of the larger survey of the cost of medical care; and Dr. Henry R. Viets, who read a paper on "The Destruction of the Hunter Manuscripts".

In the evening there was a dinner at the Maryland Club, attended by about seventy-five people, in honor of the late Dr. William S. Halsted, the pioneer surgeon of the Johns Hopkins Hospital. A bust of Dr. Halsted was presented to the Medical School. The principal speakers were Dr. Joseph C. Bloodgood, Dr. William G. MacCallum, Dr. Hugh Young and Dr. Harvey Cushing.

PHYSICIANS REGISTERED BY MASSACHUSETTS BOARD OF REGISTRATION IN MEDICINE IN EXAMINATION HELD JULY, 1929; ALSO PHYSICIANS REGISTERED UNDER NATIONAL BOARD ENDORSEMENT

National Board registrants are indicated by *, since November, 1928.

Adams, John, Jr., 704 Huntington Avenue, Boston, Mass.

Alferes, John DeF., 65 New Boston Road, Fairhaven, Mass.

Allen, Eugene E., Lowell Corporation Hospital, Lowell, Mass.

Allen, Mira May, State Farm, Mass.

Alpert, Louis, 27 Brooks Street, East Boston, Mass.

Angelo, Gaspar, 6 Oakland Avenue, Somerville, Mass.

*Ballantyne, George W., City Hospital, Worcester, Mass.

*Barr, Joseph S., Massachusetts General Hospital, Boston, Mass.
Barton, Benjamin, Chelsea Memorial Hospital, Chelsea, Mass.
Barton, Maurice Arthur, 481 Norfolk Street, Mattapan, Mass.
Barzilai, Gemma, 7 Naples Street, Brookline, Mass.
*Beetham, William P., 2 Arlington Street, Boston, Mass.
Belhumeur, Gideon A., 93 Hamlet Avenue, Woonsocket, R. I.
Benjamin, Barnard H., 585 Norfolk Street, Mattapan, Mass.
Bittle, Harry, 61 Cottage Street, Chelsea, Mass.
Bloom, Myer, Danvers State Hospital, Hathorne, Mass.
Blumenfeld, Abraham, 31 North Anderson Street, Boston, Mass.
Bombaci, Augustine, 129 Trenton Street, East Boston, Mass.
Brenner, Nathan, 78 Walnut Street, Chelsea, Mass.
Brosnan, James T., 38 Second Avenue, Lowell, Mass.
Brown, Harold F., 132 Homer Street, Newton Center, Mass.
Burke, Laurence F., 28 Clark Road, Revere, Mass.
Butts, John B., 27 Samoset Street, Ashmont, Mass.
Byrne, Joseph L., 125 Bunkerhill Street, Lawrence, Mass.
Chaya, Frank F., Box 162, Salem, N. H.
Christenson, Aileen D., Belmont Hospital, Worcester, Mass.
Clancy, Arthur W., 4 Otis Place, Newburyport, Mass.
Cohn, Samuel A., 31 Grant Place, Waltham, Mass.
Conway, James F., 1 Sachem Street, Roxbury, Mass.
Denning, Walter S., 559 Heath Street, Chestnut Hill, Mass.
Denslow, John S., 43 Evergreen Street, Jamaica Plain, Mass.
Djerf, Frederick J., 202 West Newton Street, Boston, Mass.
Dunphy, Herbert G., Boston City Hospital, Boston, Mass.
Engelbach, Albert G., Massachusetts General Hospital, Boston, Mass.
*Epstein, Samuel H., 33 Moraine Street, Jamaica Plain, Mass.
*Fish, James E., Massachusetts General Hospital, Boston, Mass.
Fletcher, John R. B., 25 Waldo Street, Manchester, N. H.
Fraser, Ernest J., 314 Newbury Street, Boston, Mass.
*Freedman, Edward P., 11 Roxton Street, Dorchester, Mass.
Garcia, Rafa el R., 11 East 116th Street, New York City, N. Y.
*George, Henry L., Jr., 9 Primus Avenue, Boston, Mass.
*Gilbert, Henry R., 1121 Great Plain Avenue, Needham, Mass.
Gilman, Bernard B., 30 North Anderson Street, Boston, Mass.
Gordon, Minerva, 324 Pennington Avenue, Trenton, N. J.
Gorman, Eugene F., Boston City Hospital, Boston, Mass.
Greene, Mildred E., 681 Main Street, Waltham, Mass.
*Gregg, Ralph, 67 Hudson Street, New York City, N. Y.
Hampton, Aubrey O., 306 Riverway, Boston, Mass.
*Harper, James D., Massachusetts Eye and Ear Infirmary, Boston, Mass.
Hartman, Samuel J., Boston City Hospital, Boston, Mass.
Hanflig, Samuel S., 100th Street and Fifth Avenue, Mt. Sinai Hospital, New York City, N. Y.
*Heath, Clark W., 368 Longwood Avenue, Boston, Mass.
Hedberg, Herbert E., 422 Columbia Road, Dorchester, Mass.
Hersey, Earl G., 10 Faxon Road, Atlantic, Mass.
*Hopkins, Edward B., 24 Washington Street, Ayer, Mass.
*Howard, Philip, Jr., Henry Ford Hospital, Detroit, Mich.
Hurwitz, David, 6 Dennison Street, Roxbury, Mass.
Jacobs, Leo D., 9 Verrill Street, Mattapan, Mass.
*Johnston, Reginald F., 5 Sudbury Road, Concord, Mass.
Kelley, Sylvester B., 124 Woburn Street, Reading, Mass.
Kelley, Thomas A., 7 Madison Street, Somerville, Mass.
Kernan, Clement F., 16 Second Street, Pittsfield, Mass.
*Knowlton, Wilson W., 109 Broad Street, Suite 301, Lynn, Mass.
Labovitz, Joseph, 43 Esmond Street, Dorchester, Mass.
*Lancaster, Harland F., 18 Elm Park, Groveland, Mass.
Leach, George V., 121 Montfort Street, Boston, Mass.
Ledoux, Alfred C., 87 Stetson Street, Whitman, Mass.
Littman, David, 96 Highland Street, Chelsea, Mass.
Lynch, George B., Jr., Boston City Hospital, Boston, Mass.
MacDougall, Ernest C., 55 Federal Street, Reading, Mass.
*Mackie, Thomas T., 103 East 78th Street, New York City, N. Y.
*Mallory, George K., 116 Longwood Avenue, Brookline, Mass.
Mansfield, Thomas R., Massachusetts Homeopathic Hospital, Boston, Mass.
*Marlow, Frank W., Jr., 1284 Beacon Street, Brookline, Mass.
Marshall, Edward J., Boston City Hospital, Boston, Mass.
McLean, Joseph A., 136 Diamond Street, Philadelphia, Pa.
Meltzer, Maxwell, 1361 Madison Ave., New York City, N. Y.
Menard, Oliver J., 60 Fenway, Boston, Mass.
Michalowski, Valarian, Massachusetts Homeopathic Hospital, Boston, Mass.
Moran, John Ed., 50 Federal Street, Greenfield, Mass.
Moran, William G., 82 East Concord Street, Boston, Mass.
Moulton, Bryant E., Judge Baker Foundation, 40 Court Street, Boston, Mass.
Murphy, William E., Massachusetts Homeopathic Hospital, Boston, Mass.
Mutty, Lawrence T., 71 Goss Avenue, Melrose, Mass.
Noehren, Arthur G., Box 693, Williamstown, Mass.
Ostrom, Hjalmar, 657 Adams Street, East Milton, Mass.
*Pallo, John, 24 Cross Street, Westfield, Mass.
Parsons, Langdon, Free Hospital for Women, Brookline, Mass.
*Pratt, Theodore C., 1175 Boylston Street, Boston, Mass.
*Rapaport, Henry, Taunton State Hospital, Taunton, Mass.

Reardon, John P., 24 Prospect Hill Avenue, Somerville, Mass.
Reece, Edward A., 560 Columbus Avenue, Boston, Mass.
Reznikoff, Leon, P. O. Box 282, Somerset, Pa.
Riseman, Joseph E., 7 Brenton Street, Dorchester, Mass.
Ritter, Benjamin, 32 Sorrento Street, Springfield, Mass.
Robinson, Charles A., Boston City Hospital, Boston, Mass.
*Rogers, Robert P., 60 Charlesgate West, c/o Dr. A. P. Rogers, Boston, Mass.
Rolett, Daniel, 1534 President Street, Brooklyn, N. Y.
Rosenfield, Barnett H., 63 Chestnut Street, Chelsea, Mass.
*Schlesinger, Monroe J., 32 Ransom Road, Brighton, Mass.
Semenenko, Helen, 333 Harvest Street, Cambridge, Mass.
Sena, Vincent W., 24 Teele Avenue, West Somerville, Mass.
Sherwin, Herbert, 1378 Pleasant Street, Fall River, Mass.
Shulman, Maurice H., Franklin Square Hospital, Baltimore, Md.
Siegel, Lewis, 43 Columbia Road, Dorchester, Mass.
Solo, Adrian, 82 E. Concord Street, Boston, Mass.
Staffier, Dominic T., 466 Saratoga Street, East Boston, Mass.
Starr, Arnold, Beth Israel Hospital, Boston, Mass.
Stetson, Richard P., New Haven Hospital, New Haven, Conn.
Stiffle, Abraham M., 75 Warren Avenue, Malden, Mass.
Tartakoff, Joseph, 6 Lamartine Place, Jamaica Plain, Mass.
Tavares, Charles M., Jr., 33 Sagamore Street, New Bedford, Mass.
Tavares, John M., 30 Ibbetson Street, Somerville, Mass.
Tivnan, Paul Edward, 74 Fenwood Road, Boston, Mass.
Umpa, Charles L., 60 Currier Street, Methuen, Mass.
*Urmy, Thomas V., Massachusetts General Hospital, Boston, Mass.
Vaccaro, Francis J., 9 Kent Avenue, Pittsfield, Mass.
Vibber, Foster L., 12 Montello Street, Worcester, Mass.
Weiss, Soma, 97 Garden Street, Cambridge, Mass.
Wells, Clinton C., 600 Pine Street, Fall River, Mass.
Wheeler, Earl B., Boston City Hospital, Boston, Mass.
*Wheeler, Ralph E., 101 Chestnut Street, Boston, Mass.
White, Charles E., 7 Pleasant Street, Stoneham, Mass.
*Whittier, Alice A. S., Hospital Cottages for Children, Baldwinsville, Mass.
*Wilkinson, Robert S., Wellesley Park, Wellesley, Mass.
*Wilson, James L., The Children's Hospital, Boston, Mass.
Wood, John L., Lawrence General Hospital, Lawrence, Mass.
Woodruff, Richard S., 19 Bartlett Avenue, Pittsfield, Mass.
*Worcester, Blandina, 186 Marlborough Street, Boston, Mass.
Zawacki, Bruno E., 69 Garfield Ave., Easthampton, Mass.
Zielinski, Edmund J., 473 Hillside Avenue, Holyoke, Mass.

EXTRACT FROM "ON EARLY SYMPTOMS" BY E. MESTER. MED. ZTCH. "THERAPIA." 1929. NR. 17.

Vomiting is usually *par excellence* an early symptom, not only in disease of the digestive tract, including the liver and pancreas but also in the case of other abdominal organs, particularly of the urogenital system. In addition, cases are not infrequently seen in which there is no other complaint. The value of the examination of the urine for urobilinogen and of the sediment is apparent. How often "silent gallstones" betray their presence; and how often does the sediment show erythrocytes when the sulphosalicylic acid test is negative. Of greater weight and seriousness are those conditions which give rise to vomiting of cephalic origin, or, more generally, all those disease conditions of the nervous system which are associated with vomiting. I have seen a considerable number of cases where a mistaken diagnosis was made, even over a period of years. So many unnecessary operations have been performed, that now I hold rigorously to the view that in any case of vomiting it is a grave omission to fail to examine the pupils, to test the function of the eye muscles and to try the tendon reflexes. A crass error is to operate for gastric crises, which may be accompanied by hematemesis and so, to the superficial observer, resemble ulcer or cancer. In lues of the base of the brain, the bradycardia may be regarded as vagotonic in origin in the absence of quickly developing cranial nerve paralysis. I know of cases in both groups in which laparotomy was performed, naturally without relief. Those in which the attending physician suspected organic disease of the stomach, were generally operated upon after, or rather in spite of x-ray examination. One patient suspected of having gallstones fortunately escaped.

It is not always a simple matter for the consultant to set forth the correct diagnosis and escape scot free, as I know from my own experience, for I was almost insulted on one occasion because, in a case of persistent vomiting, I interested myself in the kneejerk and achilles tendon reflex. But it is not only luetic changes which have to be considered. In one case a box on the ear causing extravasation because of an abnormally thin temporal bone, led to cephalic vomiting. Perhaps there is present an undetected encephalitis from an unknown cause; or a tumor or a cysticercus. Early acousticus tumor may simulate stomach or gall bladder disease for a considerable period of time. On the other hand extensive fracture of the base can occur without vomiting; in such a case it is the point of fracture that is significant.

In a considerable number of cases, inexplicable loss of weight is the only early symptom. Headache is later, with intolerable itching. In cases of inexplicable loss of weight, one thinks of tumor of the cerebrum and if the blood pressure is low of pluriglandular insufficiency. In cases where this pluriglandular insufficiency is due to tuberculosis, one finds in spite of absence of detectable involvement of the lungs, the hectic spots on the cheeks.

CORRESPONDENCE

A MEMORIAL TO WIDAL

October 21, 1929.

Editor, NEW ENGLAND JOURNAL OF MEDICINE,
Boston, Massachusetts.

Dear Sir:

The French colleagues of the lamented Widal have formed an honorary committee which proposes to perpetuate his memory by a monument, a portrait medallion for the Faculty of Medicine and the Cochin Hospital, and by the publication of a complete edition of his works.

In this project a number of Widal's admirers and friends in foreign countries have taken part. I have been invited by the French committee which consists of Professors Abrami, Bezancon, Lemierre, Valéry-Radot, Brulé and Dr. Joltrain, to confer with those in this country who have had associations with Professor Widal or who cherish his memory, for the purpose of collecting such subscriptions as we Americans may desire to give.

Widal's character and his work are such as richly deserve this recognition. I feel sure that many Americans will wish to take an active part in the work. If any considerable number of us should feel like adding their mite, no matter how small, to the fund, our contribution will be considerable. I should be very grateful for any subscriptions for this fund. Already a considerable number of admirers of Widal in Baltimore and elsewhere have contributed.

I am proposing to send an alphabetical list of all contributors together with the total sum, to the French committee. It has seemed to me unnecessary to mention the size of the contribution excepting in case it should happen to be unusually large. I hope particularly that those who desire to take part in this demonstration will not hesitate to make even the smallest contribution.

Checks may be sent to W. S. Thayer, 1208 Eutaw Place, Baltimore.

I hope greatly that we Americans may pay a worthy tribute to the memory of one whose contributions have been so notable as those of Widal.

Believe me
Yours very truly,
W. S. THAYER.

RECENT DEATHS

SMYTH—DR. PATRICK SOMERS SMYTH, a member of The New England Ophthalmological Society from Boston, died at the Phillips House, Massachusetts General Hospital, after a short illness, October 28, 1929, at the age of 52.

The son of Mr. and Mrs. Christopher Smyth of Port Hood, Nova Scotia, he was born in that town, October 26, 1877. When he had received an A.B. from St. Xavier's College he entered Harvard Medical School and took his M.D. in 1904. Then he served as house officer at the Massachusetts Eye and Ear Infirmary and henceforth devoted himself to ophthalmology, practicing in Boston. He joined the state medical society and the American Medical Association.

Dr. Smyth is survived by his widow who was Miss Irene Kennedy, a daughter and by three brothers, one of whom is Dr. Duncan Campbell Smyth of Boston, a specialist in diseases of the eye, ear, nose and throat and a Fellow of the Massachusetts Medical Society, also by four sisters.

CHARTERIS—DR. MARY ALENA CHARTERIS, a member of the Massachusetts Medical Society in Worcester from 1900 to 1927, died at Moncton, New Brunswick, October 28, 1929, aged 64.

ELKIN—DR. SAMUEL NATHANIEL ELKIN, a Fellow of the Massachusetts Medical Society, died at his home in Roxbury, October 8, 1929, at the age of 41. He was a graduate of Tufts College Medical School in 1914, was a Fellow of the American Medical Association and practiced in Roxbury.

A MEMORIAL TO DR. E. O. CROSSMAN

Doctor Edgar O. Crossman, a member of the Massachusetts Psychiatric Society, died at his home in Bedford, New Hampshire, on June 21, 1929, after a brief illness.

Doctor Crossman was born in Vermont on December 15, 1864, and graduated from the Medical School of the University of Vermont in 1887. He practised both in Vermont and New Hampshire, and served as a major in the Medical Corps of the Army during the World War. Upon his discharge from the Army, he became connected with the United States Veterans Bureau, which he served with distinction until the time of his death. He was for a time District Manager of the New England District, and became Medical Director of the Bureau in April, 1924. In July, 1926, at his request, he was transferred from Washington to Boston to become Medical Officer in Charge of the U. S. Veterans Hospital at West Roxbury. While here he also supervised the construction of the Veterans Hospitals at Northport, L. I., and Bedford, Mass. In 1928, upon the urgent solicitation of his Chief, General Hines, and at a considerable personal sacrifice, he returned to Washington as Medical Director. He was actively engaged in his official duties when stricken with his fatal illness. He was buried with full military honors in Arlington Cemetery.

Doctor Crossman will be long remembered by those who had the privilege of knowing him as a loyal, genial friend. To his patients, his memory remains as that of a devoted, faithful physician. To the country at large, he stands as one who did much to place the medical work of the Veterans Bureau upon a high plane, and who labored unremittingly to secure to the disabled veterans of the World War hospital care of the highest quality.

The Massachusetts Psychiatric Society desires to place permanently on record its sense of loss in the passing of a distinguished member and to express to Doctor Crossman's widow and son its deepest sympathy.

NEWS ITEMS

DR. ROBERT B. GREENOUGH HONORED—It has been reported that Dr. Robert B. Greenough, assistant professor of surgery at the Harvard Medical School, was elected to the newly created office of chairman of the board of directors of the American Society for the Control of Cancer on October 9.

ANOTHER MARTYR TO SCIENCE — While engaged in research work on Botulism Dr. Marie of the Pasteur Institute, Paris, contracted this disease; death resulted.

DR. DAVID P. D. WILKIE, professor of surgery in Edinburgh University, is visiting this country for three weeks. He gave the Murphy oration before the American College of Surgeons. He will also act as chief surgeon *pro tempore* at the Peter Bent Brigham Hospital, Boston, *vice* Dr. Harvey Cushing, and later as director of surgery *pro tempore* at the Lakeside Hospital, Cleveland, *vice* Dr. Elliott C. Cutler.—*Science.*

DR. MORRIS FISHBEIN WILL SPEAK IN BOSTON—Dr. Morris Fishbein, Editor of the *Journal of the American Medical Association*, will speak in the Amphitheater of the Beth Israel Hospital on Thursday, November 21, 1929, at 8:15 P. M.
Subject: The Cost of Medical Care.

REPORTS AND NOTICES OF MEETINGS

THE HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the amphitheatre of the Peter Bent Brigham Hospital on the evening of November 12 at 8:15. There will be a presentation of cases and a paper by Dr. Cecil K. Drinker on "Lymphatics and Lymphatic Drainage."

WILLIAM P. MURPHY, M.D.

MASSACHUSETTS GENERAL HOSPITAL

A Clinical Meeting of the Staff of the Massachusetts General Hospital, will be held in the Moseley Memorial Building, on Thursday, November 14, 1929, at 8:15 P. M.

PROGRAM

Subject: Cerebro Spinal Meningitis.
1. Bacteriology. Dr. Benjamin White.
2. Pathology. Dr. Tracy B. Mallory.
3. Incidence and Differential Diagnosis. Dr. James B. Ayer.
4. Clinical Aspects. Dr. Charles F. McKhann.

Doctors, medical students and nurses are cordially invited to attend.

COMMITTEE ON HOSPITAL MEETINGS.

WINTER TRAINING COURSE FOR MEDICAL RESERVE OFFICERS

The next meeting of the Medical Department Reserve Officers of Boston and vicinity, will be held at The Kenmore, 496 Commonwealth Avenue, Boston, at 8:00 P. M., on November 13, 1929.

All Medical Department Reserve officers are urged to make every effort to attend.

Light refreshments will be served at the termination of the meeting.

F. W. WEED,
Lieut. Col., Medical Corps, U. S. Army.

CLINICAL STAFF OF THE BOSTON DISPENSARY

The November meeting of the Clinical Staff of the Boston Dispensary will be held Tuesday evening, November 19, 1929, at 8:00 P. M. at 25 Bennet Street, under the auspices of the Division of Research.

The following program will be rendered:

1. "The Diagnosis of Nephrosis." Robert W. Buck, M.D.
2. "The Significance of Axis Deviation in the Human Electrocardiogram." Samuel H. Proger, M.D., David Davis, M.D.
3. "The Occurrence of Normal Blood Pressure Readings in Essential Hypertension." David Ayman, M.D.
4. "The Value of a New Serum Test in Neurosyphilis." Arthur Berk, M.D.

Discussion: Doctors W. Richard Ohler, Paul D. White, Harry C. Solomon, James P. O'Hare and G. Philip Grabfield.

Physicians are cordially invited to attend.

AUGUSTUS RILEY, M.D., *President*,
JOSEPH J. SKIRBALL, M.D., *Secretary.*

THE ANNUAL MEETING OF THE MASSACHUSETTS PSYCHIATRIC SOCIETY

The annual meeting of the Massachusetts Psychiatric Society was held on October 30 at the Hotel Somerset and was attended by fifty-five members and guests. The speaker of the evening was Dr. Milton C. Winternitz of the Yale School of Medicine, who gave a most stimulating and interesting presentation of the origin and the purposes of the "Institute of Human Relations" which has just been organized in Yale University.

The following officers were elected:

President: Dr. L. Vernon Briggs, Boston.
Vice-President: Dr. George L. Wallace, Wrentham.
Secretary-Treasurer: Dr. Winfred Overholser, Boston.
Councillors: Drs. Douglas A. Thom and E. Stanley Abbott, Boston.

A memorial to the late Dr. Edgar O. Crossman was adopted.

WINFRED OVERHOLSER, *Secretary.*

JOINT MEETING AT NORTH READING STATE SANATORIUM

An interesting meeting of the four District Societies, Essex South, Middlesex North, Middlesex East and Essex North, was held on Wednesday, October 16, 1929, at the Sanatorium with 115 present. It was a beautiful New England Fall day.

From 2-4 there was an inspection of the institution in groups, under supervision of the Superintendent, Carl C. MacCorison, M.D., and Staff. There are in the institution two hundred tuberculosis patients, all children, between three and seventeen years. All such are given semi-weekly physical instruction, by competent instructors, as soon as the condition warrants. Included in this practice is a certain amount of military drill. A swimming pool is provided during the suitable months and is supervised. All children, when able and of school age,

attend one session of school under instruction of Normal School graduates.

A new brick structure is being completed with 30 beds, to be used as an isolation hospital, in which all new cases are to be kept for 28 days, thus protecting against contagion being brought to the Sanatorium proper.

Individual ear-phones attached to radios are supplied in the wards and all patients appeared contented and happy.

At 4 P. M. an address of welcome was given by George H. Bigelow, M.D., State Commissioner of Public Health, and he urged physicians to continue to use great care in the diagnosis and treatment of early tuberculosis, so that every case of this disease may be cured.

Thomas J. O'Brien, M.D., of Boston, Executive Assistant to the President of the Massachusetts Medical Society, spoke interestingly of the work of the Society in protecting the citizens of the Commonwealth from the disastrous effects of the activities of the various cults and healers, wholly ignorant of the structure of the human body and the diseases and disabilities affecting the body and mind. Great regret was expressed at the failure to secure the adoption of the bill requiring the extension of compulsory vaccination against smallpox, which passed the House and Senate last spring.

At 4:30 an hour's interesting clinic was presented by the Staff, including graphic x-rays of numerous cases of tuberculosis, and showing clearly a progress in the right direction concurrent with the improvement of health of the patient.

Clarence A. Bonner, M.D., Superintendent of Danvers State Hospital, spoke for one hour in an instructive manner upon his subject, "Early Diagnostic Points in the Commoner Mental Diseases, of Value to the General Practitioner." He outlined particularly the symptom-complex, frequently found in cases of dementia praecox, and which now forms about 45% of all admissions to his Hospital.

An excellent supper was served at 6:30, during which votes of thanks were extended to the Staff and to the speakers.

A good time was enjoyed by all.

Thomas L. Jenkins, M.D., of Topsfield, President of Essex South District Medical Society, presided at the meeting.

J. FORREST BURNHAM,
Secretary Essex North District Society.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

1929-1930—Massachusetts Dietetic Association. Complete schedule appears on page 852, issue of October 24.

November 12—The Harvard Medical Society. Complete notice appears on page 964.

November 13—Winter Training Course for Medical Reserve Officers. Complete notice appears on page 965.

November 14—Massachusetts General Hospital. Detailed notice appears on page 964.

November 19—Clinical Staff of the Boston Dispensary. Detailed notice appears on page 965.

November 21—New England Heart Association. Complete notice appears on page 906, issue of October 31.

December 2-6—The Radiological Society of North America. Complete notice appears on page 149, issue of July 18.

February 10-14, 1930—American College of Physicians. Complete notice appears on page 853, issue of October 24.

March-October, 1930—International Medical Postgraduate Courses in Berlin. Complete notice appears on page 853, issue of October 24.

May 5-10, 1930—The International Congress on Mental Hygiene. Detailed notice appears on page 853, issue of October 24.

September 4-8, 1930—Fifth International Congress on Physiotherapy. Complete notice appears on page 906, issue of October 31.

DISTRICT MEDICAL SOCIETIES

Essex North District Medical Society

November 7 — Thursday — Censors' meeting at Hotel Bartlett, 95 Main Street, Haverhill, (Telephone 3430) at 2 P. M. sharp.

January 8, 1930—Wednesday—Semi-annual meeting at Centre Church Vestries, Main Street, corner of Vestry Street, opposite City Hall, Haverhill, (Telephone 548) at 12:30 sharp.

May 1—Thursday—Censors' meeting at Hotel Bartlett, 95 Main Street, Haverhill, (Telephone 3430) at 2 P. M. sharp. Candidates should present their diplomas to the Secretary one week in advance.

May 7—Wednesday—Annual Meeting.

J. FORREST BURNHAM, Secretary.

Essex South District Medical Society

Thursday, November 7, 1929—Censors meet for Examination of Candidates at Salem Hospital, at 3.30 P. M.

Wednesday, December 4, 1929—Salem Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Howard B. Sprague, of Massachusetts General Hospital Heart Clinic. Subject: "Recent Progress in the Diagnosis and Treatment of Heart Disease." General Discussion from the floor.

Wednesday, January 8, 1930—Beverly Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker and Subject to be announced later.

Wednesday, February 5, 1930—Council Meeting, Boston.

Wednesday, February 12, 1930—Danvers State Hospital, Hathorne. Clinic 5 P. M. Dinner 7 P. M. Speakers: Dr. Winfred Overholser. Subject: "Criminology and Psychiatry." Mr. Clark, District Attorney for Essex County, who will speak on the legal aspect.

Wednesday, March 5, 1930—Lynn Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. R. S. Titus, Boston. Subject: "Some Aspects of Caesarean Section," illustrated with Moving Pictures. General Discussion from the floor.

Wednesday, April 2, 1930—Hotel Hawthorne, Salem. Dinner at 7 P. M. Speaker: Dr. Timothy Leary, Boston. Subject: "Diagnostic and Therapeutic Pitfalls in Intracranial Pyogenic Infections."

Thursday, May 1, 1930—Censors meet for Examination of Candidates at Salem Hospital at 3:30 P. M.

Tuesday, May 13, 1930—Annual Meeting. The Tavern, Gloucester. Speaker: Dr. C. Macfie Campbell, Director of Boston Psychopathic Hospital. Subject to be announced later. Ladies invited. Dancing.

R. E. STONE, M.D., Secretary.

Franklin District Medical Society

The meetings of the Franklin District Medical Society will be held at the Weldon Hotel, Greenfield, Mass., on the second Tuesdays of November, January, March and May, at 11 A. M.

CHARLES MOLINE, Secretary.

Hampshire District Medical Society

The meetings of Hampshire District will occur at 11 A. M. the second Wednesdays of December, February, and April and will be held at the Dickinson Hospital, Northampton. The annual meeting will be held in May.

LUTHER O. WHITMAN, M.D., Secretary.

Middlesex East District Medical Society

November 13—At Reading.
January 15—At Boston, Harvard Club.
March 12—At Melrose.
May 13—At Unicorn Country Club.

ALLAN R. CUNNINGHAM, Secretary.

Middlesex South District Medical Society

April 16, 1930—Annual meeting. Commander Hotel, Cambridge. Program to be announced.

Winter meetings—jointly with Suffolk District.

May, 1930—Censors' meeting.

ALEXANDER A. LEVI, Secretary.

Norfolk District Medical Society

November 25—Roxbury Masonic Temple. Dr. Roger Graves, "Urology from the Standpoint of the General Practitioner." Surgical discussion. Medical discussion.

January 27, 1930—Roxbury Masonic Temple. "Recent Advances in Diagnosis and Treatment of Syphilis." The speakers will be Drs. C. Morton Smith and Austin W. Cheever.

February 24, 1930—Roxbury Masonic Temple, 8:00. Dr. Edwin H. Place has been invited to read a paper and has been given carte blanche in the selection of his subject.

March 24, 1930—Roxbury Masonic Temple. Dr. Burton E. Hamilton. Subject to be announced.

May 6, 1930—Annual Meeting. Program to be announced.

The Censors meet for the examination of candidates, November 7, 1929, and May 1, 1930, in the Roxbury Masonic Temple, 171 Warren Street, Roxbury, at 4:00 P. M.

Applications must be in the hands of the Secretary at least one week previous to date of examination.

Owing to inability to engage the Roxbury Masonic Apartments on the last Tuesday of the month, such meetings as will be held in the Roxbury Temple will be held Monday evenings as per the dates above.

FRANK S. CRUICKSHANK,
Secretary, Norfolk District Medical Society.

Suffolk District Medical Society

Censors' Meeting. The Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, November 7, 1929, at 4:00 o'clock.

LELAND S. McKITTRICK, Secretary.

205 Beacon Street, Boston, Mass.

BOOK REVIEWS

Diseases of the Stomach. A Text-book for practitioners and students by MAX EINHORN, M.D., Emeritus Professor of Medicine at the New York Postgraduate Medical School and Hospital; Consulting Physician to the Lenox Hill Hospital, New York. Seventh Revised Edition. New York. William Wood and Company. 1929. Price $6.00.

This seventh edition of a well-known work is a careful exposition of the subject of diseases of the stomach including the special methods, many of them original with the author, employed in diagnosis and treatment. After a brief chapter on the anatomy and physiology, a long second chapter on methods of examination describes, in addition to interrogation of the patient and physical examination of the ordinary sort, numerous auscultatory signs, oesophagoscopy, gastroscopy, gastrodiaphany, roentgenography, radium transillumination, and many tests of gastric contents. Chapter III is a relatively brief discussion of diet. Chapter IV on the local treatment of the stomach describes lavage and gastric spray, the stomach powder blower and electrization. The remaining three-quarters of the book discusses the separate diseases and disorders of the stomach, dividing them into 1. Organic diseases with constant lesions, 2. Functional diseases with variable lesions, 3. Abnormal conditions with reference to the size, shape, and position of the stomach, and 4. Nervous affections. The organic diseases include acute and chronic gastric catarrh together with phlegmonous and toxic gastritis, ulcer, erosions of the stomach, and cancer. The functional diseases comprise conditions typified by abnormal findings in the gastric contents from hyperchlorhydria to achylia gastrica with, in addition, a chapter entitled *Ischochymia* which turns out to mean undue stagnation of chyme in the stomach whether from pyloric stenosis or from any other cause. In the third division enteroptosis is the chief subject. The nervous affectations described appear under presenting symptoms such as bulimia, hyperaesthesia, eructation, etc., together with spasm of the cardia and of the pylorus, and nervous vomiting. These descriptions represent the result of the efforts of modern diagnosis to classify all abnormalities of the stomach into clinical entities. In this respect the specialist in stomach diseases goes further than do most practitioners of internal medicine, and undoubtedly in many cases to the advantage of the patient. The reviewer, however, is unable to suppress the opinion that the functional disorders of the stomach are too variable in individual cases to be susceptible of such rigid classifications as this book attempts, and that we must still await a more fundamental knowledge than is yet attained before our command over the symptoms of dyspeptic patients can be satisfactory.

Principles of Chemistry: An introductory textbook of inorganic, organic and physiological chemistry for nurses and students of home economics and applied chemistry, with laboratory experiments. By JOSEPH H. ROE, PH.D. Second edition. C. V. Mosby Company, 1929. 427 pp. Price, $2.50.

This is an excellent book, well and simply written and containing a surprising amount of information; at the same time the fundamental principles of the science receive adequate consideration.

The author has introduced into this revision "a chapter upon the chemistry of the blood, in which those phases of blood chemistry that the nurse should know have been stressed". It would seem even more appropriate to have devoted more attention to the chemistry of some of the drugs which the nurse will constantly see in the hospitals; a number of the drugs included in the "Useful Drugs" of the American Medical Association could have been discussed with profit. In some cases the official names should have been used instead of proprietary names.

However, the work is an admirable elementary presentation of the modern science of chemistry and a book, the reading of which many physicians who may have neglected a science of such great importance in medicine, might find interesting and profitable.